

**RADIOLOGICAL EVALUATION OF PRIMARY
TUBERCULOSIS IN MANTOUX
POSITIVE CHILDREN**

**THESIS
FOR
DOCTOR OF MEDICINE
(RADIO-DIAGNOSIS)**



**BUNDELKHAND UNIVERSITY
JHANSI (U.P.)**

" I that am curtailed of this fair proportion,
Cheated of features by dissembling nature,
Deform'd unfinish'd, sent before my time,
Into this breathing world, scarce half made up,
And that so-lamely and unfashionable
That does bark at me as I halt by them".


Richard (Shakespeare)

C E R T I F I C A T E

This is to certify that the work entitled "Radiological Evaluation of Primary Tuberculosis in Mantoux Positive Children", which is being submitted as Thesis for M.D. (Radiodiagnosis) examination, 1991 of Bundelkhand University, Jhansi by PRAVEEN KUMAR JAIN, has been carried out under my supervision and guidance in the department of Radiology. The techniques embodied in the thesis were undertaken by the candidate himself and the observation recorded have been regularly checked by me.

He has put in necessary stay in the department according to University regulations.

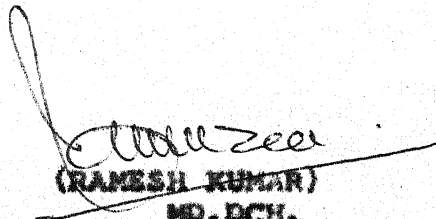
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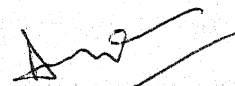
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INTRODUCTION

INTRODUCTION

THE CHALLENGE :

The battle of the human race with tuberculosis dates back to antiquity. Historians are divided on the questions of the exact date and the mode of the first encounter. Certain pre-historic skeletons, a few preserved bones and joints in the planes of the Gangas, show pathological changes which are evidently tuberculous. Inscriptions on a few tablets found in Babylon contain references to the disease. A few mummies of Egypt show conditions simulating tuberculosis. All these evidences go to show that practically no ancient civilization was spared off the evil touch. Hippocrates gave the first description of the disease, recognised as "phthisis" at the time. Aristotle recognized the infectious nature of the disease, and wondered why a person in contact with the sufferer developed the disease, while a person in contact with dropsy did not do so.

In 1882, Robert Koch discovered the causative organism, the tubercle bacilli. Till that time it was almost a guerilla warfare. As man's attack in those days was at best a grope in the dark, it could not hit the target. Consequently, the casualties on the side

were quite high. The weak ones on his side succumbed in large numbers; those in better conditions were badly maimed; the fittest, of course, survived. Koch's momentous discovery led to further advances in the knowledge of the disease. Man acquired quite a number of lethal weapons against the dreaded enemy. The results were spectacular. Tuberculosis came down in rank in the list of the leading causes of death. But economically under - developed countries could not keep pace with those leading to failure, in the control of the disease.

Even in the present times, with all the modern knowledge man possesses, the disease exerts a profound influence on the morale and economy of every nation. The repercussions it produces, though chronic and less dramatic than the plague or the typhus, are no less severe. Ill health restricts working time, lowers productivity and calls for expensive medicines, and thus adversely effects national prosperity. This is the story in the usual course of life. But during wars and the following years of hardship, the problem assumes gigantic proportions as the disease takes a great toll of lives even in modern time. During famine and floods, when the native resistance of man crumples, the disease reaps a rich harvest. When measles or other debilitating diseases appear, tuberculosis

lurking in the body, joins hand with them and together have their hey day. The latest weapon in the enemy's armamentorium are resistant strains and mutants. Certain atypical mycobacteria have also made their appearance. Their pathologic significance is still in the realm of conjecture.

Thus it is evident that man has suffered too long and too severely at the hands of this pestilence. Even now the enemy appears to be opening up new fronts. Hence a concerted effort has to be made from all fronts to control the disease.

The concepts of tuberculosis control have undergone a radical change in recent times. Previously it was thought that the primary infection conferred immunity on the individual and hence was considered a benign condition. Now it is increasingly recognised that primary infection, though innocuous in a majority of cases, is still a potential source of danger. So, from caring for the sick and the dying, the attention has been shifted to a search for the unrecognised cases among the apparently healthy. Early detection and appropriate management of all primary disease is the cornerstone of recent tuberculosis control programme.

For this, a thorough understanding of the various clinical manifestations and epidemiological and bacteriological aspects of the primary disease is essential. In the western countries and the U.S.A., innumerable studies were carried out. But the findings vary from place to place. This is especially true in India, where, as Dr. Bogen (1960) has remarked, the tuberculosis problem presents certain anomalies. According to him, findings here "contradict the lessons laboriously learned from previous experiences in other countries".

The various clinical and roentgenological manifestations are well seen in the young children; in adults, the clear cut picture is seldom seen. Hence this study was undertaken to demonstrate the epidemiological and bacteriological aspects of the disease as applied to the conditions obtaining in our surroundings. The frequency of the various clinical manifestations, as seen in our hospitals, are expected to be demonstrated by this study. As this is only a short term study (One year) one can only show the immediate prognosis of the primary infection. It is a well known fact that children below the age of 5 years are more prone to the

infection than an older age group. Hence this age group has been selected. Broadly speaking, the extent of the tuberculous infection in children is a reflection of the disease in adults. Recent surveys have shown that 40% of India's population is constituted of children below the age of 15 years. In that context, a study of the disease process in children will have a definite significance.

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## REVIEW OF LITERATURE

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## REVIEW OF LITERATURE

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"Tuberculosis may have been the first-born of the mother of pestilence and disease. It evidently existed in the pre-historic time on the planes of Gangas as manifested by pathological conditions found in preserved bones and joints", (quoted from Tuberculosis - J. Arthur Myers). From the dawn of history, on through the centuries, a glimpse is caught here and there of the presence of this disease in people and animals. In the long history and amid the large literature of tuberculosis, certain dates and classic publications are outstanding, signifying major advances in knowledge and understanding of this disease.

In the Rig Veda (6000 B.C.) there are observations concerning the behaviour of the tuberculosis in people. This is also true of Egyptian papyri and ancient Chinese writings. Evidence of Pott's disease dates back to Neolithic period (5000 B.C.). A number of bodies mummified between 2000 and 1000 B.C. have been found with evidence of tuberculosis of bones and joints, indicate that man has been affected with tuberculosis since ages. Charak and Sushruta (500 B.C.) had mentioned about the disease in their writings. Aristotle (450 B.C.) recognised its contagious nature.

Hippocrates (470 - 376 B.C.) named it "Phthisis" for the first time, meaning thereby to waste away. It has been given other names such as "captain of the man's death" and the great white plague", *Spes Phthisis* referred to the eternal hope of the phthisised person, even though in the last stage of the disease. The word "tuberculosis" was coined for the first time by Scholein (1839). Kortum (1789) apparently, was the first to attempt to transmit the disease from human to human. Klenche (1843) transferred tubercles to a rabbit but apparently did not clearly understand the significance of his experiment.

Parrot (1876) in a short impersonal account, reported his observations on the relationship of the pulmonary tuberculosis in childhood to tuberculosis, of the tracheo bronchial glands. He thought that the tracheo bronchial glands were the mirrors of the lungs and conversely there is no bronchial adenopathy which does not have it's origin in the lungs. But Laennec said that the bronchial glands were often tuberculous at the same time as the lungs were healthy.

Robbert Koch announced the discovery of the causative organism *Bacillus tuberculosis*, on 4th April,

1882. He isolated the bacterium, grew it on artificial media, inoculated into the animals and produced the same disease. Koch later noted the difference in the reaction complex produced by the inoculation of the organism in a healthy animal "previously uninfected" and one in a "previously infected". This is known as 'Koch's phenomenon'. It is the basis of the concept of 'Primary' and reinfection tuberculosis.

In 1890, Koch announced a curative agent against tuberculosis and named it as 'tuberculin'. He noted the symptoms (local and general) produced by a subcutaneous injection in a tuberculous patient, but he paid no importance to the reaction produced at the site of injection. Epstein and Escherich for the first time mentioned diagnostic importance of the reaction produced at the site of injection. With the advent of the phenomenon of allergy it was established that tuberculin allergy means an individual who had experienced a specific body change through infection with the living or dead tubercle bacilli. Soon after this, various tests came into picture for detecting the tuberculous sensitivity.

CHILDHOOD TUBERCULOSIS :

In 1912, thirteen years after the discovery of tubercle bacillus came Ghon's classic monograph, "Der Primair Lungenherd bei der Tuberculose der Kinder" in which the primary lung lesions and the relationship of the glands were described. Ghon also mentioned extra pulmonary primary lesions on skin and on mucus membranes where the same relationship of primary focus and regional adenitis was demonstrated and he confirmed Parrot's work.

In 1916, Ranke postulated three stages in tuberculous infection associated with different degrees of allergy; the first that of primary infection; the second that of dissemination and hyper-sensitivity and a tendency to exudative lesions, while the third was the stage of increased resistance and lowered sensitivity and localisation of infection. This broadly speaking, indicates the stages of tuberculosis after primary infection.

The next masterly study was the series of papers by Rich and McCordock (1929) in which they considered the relationship of host resistance, virulence of the organism, allergy and the size of the infecting dose in determining the character of the resultant lesions. They advanced and clarified concept of the differences of the progressive adult and childhood tuberculosis.



In France, the "Grancher" system of care for the separation and protection of infants in contact with infective parents was evolved from 1907, and in 1920 B.C.G. vaccine was given its first clinical trials, the results which caused so much controversy that the acceptance of its usefulness was delayed in many countries.

Wallgren and his colleagues (1925) published many studies of tuberculosis in childhood. During this time they also established suspicion of children in contact with infection and used B.C.G. vaccination as a method of protection. They emphasised the relationship of erythema nodosum and primary infection.

Rich (1944) published a book "The Pathogenesis of Tuberculosis" in which he has described a critical survey of contemporary knowledge concerning the relationship of tubercule bacillus and human host and the basis factors which cause variations in that relationship. Brailey and Hardy (1958) published a clinical and epidemiological study of 437 White and 492 Negro children admitted for observation between 1928 and 1944 and supervised until 1950. This dealt with the prognosis of the primary infection and with the risks of developing progressive adult type pulmonary disease before the introduction of the chemo-therapy.



Hardy (1958) has noted "Primary tuberculosis" is the result of primary tuberculous infection in an individual often a child, though sometimes an adult, who having no previous experience with this type of infection has, acquired neither resistance nor previous hypersensitivity towards it. He has further noted "very rarely a second primary infection may occur in an individual, who has lost the immune responses, resulting from the first infection". Neaf (1959) has clearly noted "the term primary tuberculosis should be limited to the lesion which arises from the infection that takes place prior to the development of tuberculin sensitivity".

In South India (Saidapet), 30,000 population was tuberculin tested and prevalence of the tuberculosis was found to be 2.3% (Benjamin et al, 1939). A tuberculin survey was conducted in Uttar Pradesh (1949-51) under the auspices of W.H.O. This revealed a higher reaction rate in urban population as compared with rural population. In industrial cities 73% of people were tuberculin positive by 15 years of age.

Sarin et al (1957) carried out histologic studies of hepatic lesions in 100 cases of pulmonary tuberculosis in which they reported tubercle in 15, focal cellular collection in 35, focal necrosis in 47, fatty metamorphosis in 27 and reticulo-endothelial hyperplasia in 57 cases.

Bently and Grybowski (1954) reported that out of 317 children with uncomplicated primary complex 115 had a paranchymal faccus and 202 had only lymphadenopathy.

In 1958 Walker, in a study of 538 children, primary complex was seen in 280 cases (mottling with hilar adenitis), primary cavitation in 5, bronchopneumonia in 3 and miliary tuberculosis in 60 cases.

Manchanda et. al. (1966) reported their observation on 225 children, 43 were found to have primary complex, 86 were found to have lymphadenopathy, collapse were in 4 cases, consolidation in 11 cases, bronchopneumonia in 2 cases, pleural effusion in 5 cases, middle lobe syndrome in 2 and cavitation formation in one case.

Dinglay (1966) conducted a study and found that out of 500 cases of primary tuberculosis 260 had glandular enlargement and 28 cases had primary complex, consolidation in 170 cases, collapse in 37 cases, pleural effusion in 56 cases and calcification in 5 cases.

In 1976, A. Govindan and R. Narmada conducted a study on 1500 children, clinically suspected primary tuberculosis. There detailed history was taken. All the children were tested for montoux positivity and their chest X-ray was done.

The various features observed on the chest roentgenograms were numbered and tabulated in table as follows -

TABLE

Showing various features of primary tuberculosis  
in Chest X-ray  
&  
various Roentgen manifestation of primary  
complex

1. Primary focus with perifocal inflammation
2. Lymphangitis
3. Hilar adenitis
4. Mediastinal adenitis
5. Inter lobar pleural opacification
6. Primary focus calcification
7. Calcification of lymphatics
8. Glandular calcification
9. Pleural effusion
10. Pleural thickening
11. Pleural calcification
12. Atelectasis
13. Bronchiectasis
14. Miliary tuberculosis
15. Bronchopneumonia
16. Associated carries of thoracic spine
17. Tubercular pneumonia
18. Obstructive emphysema
19. Extra pulmonary calcification
20. Cavitation.

Kotaiah (1958) published his study of primary pulmonary tuberculosis in 195 cases at Visakhapatnam. He observed that highest incidence of tuberculosis in children seem to be in higher age group (10-15 years) than in those published by Western countries and there were only 23 cases (12%) below the age of three year, 98% of his cases were Mantoux positive. In this series, mediastinal lymph node enlargement was seen in 50% cases. Manchanda et al, (1958) reported 215 cases (15.7% of 5366 cases) from Amritsar. They had a history of contact in 30% of the subjects, symptoms were vague but gastrointestinal symptoms were most common. This series had higher incidence in 5 - 10 year age-group.

Udani (1960) in his autopsy report of 100 children between the age of two months and ten years revealed that tuberculosis of various types formed 19.8% of the total cases on clinical side and tuberculosis was major cause of death in 10% of cases, though in 26% of cases, there were various types of tuberculous lesions as associated lesions.

Barucha et al, (1961) reported one case of miliary tuberculosis with granulomatous lesions in the liver. Udani (1962) reported 8 cases with hepatic and splenic lesions in 36 tuberculous children, who were subjected



to autopsy. Three of them had miliary tubercles of liver as a part of generalised miliary tuberculosis, whereas in one case the miliary tubercle in the liver were realised as an abdominal picture of miliary tuberculosis; in others there was a picture of caseating tuberculoma, toxic hepatopathy, cirrhosis and amyloidosis. Balkrishnan and Sharma (1962) reported 6 cases of tuberculosis in whom they studied hepatic lesions.

Prakash et al, (1963) observed 273 tuberculin positive children at Lucknow and found the incidence of 10.8%, 32.6% and 40.7%, among 0-3 years, 3-7 years and 7-12 years age-groups respectively. In their series 35% cases had a positive history of contact and on radiography, 84.2% of the cases showed intra-thoracic abnormalities.

In 1964, Reddi et al studied hepatic lesions in 20 tubercular children and found that 80% (4 out of 5) of miliary granulomatous lesions were seen in cases of miliary tuberculosis, the remaining was seen in tuberculous meningitis and none was seen in other forms of tuberculosis.

In 1966, Ramchandran and Purnayyan from Thanjavur, presented an analytical study of 365 children out of which 321 (87.9%) were P.P.D. positive, but the rest of the cases (44) were also suffering from tuberculosis. They found that

maximum incidence was amongst 0-2 years age-group (46.8%) but only 10% gave the history of contact. In this series, the commonest radiologic finding was mediastinal glandular enlargement, the classic primary complex was seen only in 32 radiographs out of 347.

Classifications : On the basis of the detailed studies of the type and duration of the disease in children and adult, Sekulick (1955) described two types of tubercular processes.

- a) Primary tuberculosis : as benign - mostly affecting children.
- b) Secondary of reinfection type - mostly affecting the adults.

Primary Tuberculosis :

This is the form of the tuberculous process which develops in an individual who having had no previous exposure with this type of infection has acquired neither resistance nor previous hyper-sensitivity towards it. It is more common in infants and children because they provide a "virgin soil" more frequently than the adults. 'Primary focus' is the structural change or reaction of the tissues brought about by the tubercle bacillus at the site of first recognisable implantations. The term 'Primary adenitis' is



employed for the tuberculous adenitis associated with primary focus. The first affected node is known as 'primary node' and the others as 'satellite node'. The primary focus the lymphangitis and the regional adenitis are known as the primary tuberculous complex. Primary tuberculosis can lead to seriousness and crippling disease in children, it can be fatal and is considered as 'fountain head' of clinical tuberculosis of adult life.

#### Incidence :

In Vienna, Hamberger and Monti (1909) found that every child before reaching adult life was infected with tuberculosis and it is likely that in all the cities of Europe, the age incidence of the infection at that time was much the same although the comparative incidence in country bred children was less known. The incidence in India has been quite high since long and even today. Ukil (1931) reported it as 11.40% for 0-5 years, 30 percent for 5-10 years and 33.3% among 10-15 years age-groups. Similarly, other surveys done in localised areas have also reported a very high incidence of the disease (Frimodt Moller, 1948; B.C.G. Tean, 1951). Chatterjee (1957) and Basu et al (1958) reported moderately high incidence of tuberculous infection in sick children attending the

children hospital and centre. Extensive work done by Primodt-Moller (1962) at Madanpelle, a rural area in Southern India revealed that 95% of this population react to a dose of 100 T.U. (with reactions of 5 mm. or more) and that young children appeared heavily infected, at the age of 5 years 60% and at 10 years 85% react to 100 T.U. A systemic programme of case finding, X-ray and tuberculin testing and hospitalization of the infectious cases reduced the mortality in that area from 200 to 21 per 100,000 in less than four years duration. Udani (1962) has quoted in his article 'Management of tuberculosis in children that a sample survey of the population of Bombay city, tuberculous disease was found in 5.5%. The incidence is likely to be higher in the population of low socio-economic groups who live in overcrowded, ill-ventilated one room tenements in the chawls of the city.

However, the incidence is falling in so called better developed countries. Miller (1958) has stated that in 1957 in London, about 2% of children were tuberculin positive in 1st year, 10% at 10 years, 25% at 14 years whereas in 1939 it was 16.8% at 4 years, 26.4% at 8 years and 47.2% at 12 years (Bradshaw). In United States, for

the kindergarten children it has been estimated as 1.5% and 2.6% respectively for White and Negro children (Maha, 1963).

Mode of infection :

There are two main types of primary tuberculous infection, the air borne and the alimentary, and the former is more frequent. It is active in 90% of all infection; in countries with no bovine tuberculosis, the percentage is 100. There is no general agreement as to whether infection by droplets or by dust has the dominant role.

In addition, direct implantation of tubercle bacilli into the skin or mucus membrane may occur in exceptional cases. By congenital tuberculosis is meant a foetal infection passing across placenta and via the umbilical vein, this is a rare phenomenon.

Incubation period :

The end of the incubation period is marked by the appearance of tuberculin sensitivity. The length of pre-allergic period varies considerably in different cases the reasons for this variability include intensity and virulence of the infection, the age of the child and individual variation of reaction. In spontaneous natural infection in man the pre-allergic period has been found

to be not less than 3 weeks and not more than eight weeks and is generally 5-6 weeks (Wallgren, 1941; Waz-Hockert, 1947).

Tubercle Bacilli and the Host :

Tubercle bacilli (*Mycobacterium tuberculosis*) are characterised by their capacity to produce the infection or disease known as tuberculosis, in susceptible animals. They are acid fast and an aerobic. There are five types of tubercle bacilli, human, bovine, avian, murine and cold blooded. They are defined by their pathogenicity for different species of animals. *Micobacterium* contains 25% lipid (dry weight). The polysaccharides of tubercle bacilli have arouse interest by their antigenic activity but the antibodies which have been demonstrated are of little value in diagnosis and have no relationship to immunity. During the growth of tubercle bacilli proteins are liberated which are concerned in tuberculin sensitivity although nothing is known of their function in the organism.

The relationship between the tubercle bacilli and the human host is complex. The outcome of their encounter presenting a wide spectrum of possibilities. There are many examples of nurses caring for patients with advanced open chronic disease over many years, who



never show any clinical or radiological evidence of infection and have a persistently negative tuberculin test. Course of this infection is variable - one person develops only a small limited primary complex which never gives rise to illness, whilst another rapidly develops extensive progressive disease.

#### Forms of Primary Tuberculosis :

It has been estimated that about 95% of the lesions of primary tuberculosis are intra-thoracic, out of which pulmonary form is commonest. The other forms ranking next to pulmonary, in order of frequency, are gastro-intestinal, primary adenitis, tonsils, skin and congenital tuberculosis etc.

#### Primary Pulmonary Tuberculosis :

The lung is the commonest site for primary infection. The primary focus is usually sub-pleural and without any strong predilection for any special segment or lobe. Anatomical distribution with order of frequency are : right upper lobe, right lower lobe, left upper lobe, left lower lobe and finally the middle lobe. This is seen in children without any religion, Sex or racial discrimination.



### Clinical Features of Primary Pulmonary Tuberculosis :

The manifestations depend on the type of primary disease. In simple primary pulmonary tuberculosis, the cases usually are asymptomatic or show minimal or a non-specific illness. Wallgren (1948) is of the opinion that fever is the most common clinical manifestation of primary tuberculosis infections. Probably all infected children display an increased body temperature, which is in no way characteristic of tuberculosis. The other manifestation like pulmonary pleural effusion, segmental lesions and hematogenous forms are almost always associated with characteristic symptoms in association with the constitutional symptoms i.e. cough anorexia, prostration, malaise, night sweating, loss of body weight, failure to thrive etc. The signs of pulmonary primary infection are largely constitutional and signs or symptoms directly referable to the lungs are uncommon except in young infants and then if pulmonary symptoms are present the condition has usually spread beyond that of the simple primary complex.

### Pathogenesis and Course of Primary Complex :

At the site of initial focus, e.g. in the paranchyma of the lung, there is at first an accumulation of polymorphonuclear leukocytes. This reaction is

temporary and is followed by proliferation of epitheloid cells, which surround the tubercle bacilli, creating the typical tubercle formation. The tubercles are usually surrounded by an accumulation of lymphocytes, and giant cells are usually present. Tubercles may remain discrete or may become confluent; central caseous necrosis is commonly present.

In the majority of the people caseation in primary focus rarely exceeds a centimeter in diameter and as the infection is contained by the host, healing begins and capsule develops as fibroblasts and lymphocytes appear at the periphery and collagen fibres are laid down around the focus. The tendency of the primary lesions both in paranchyma and nodes is towards healing in the majority of instances.

The various possibilities are - (i) healing, (ii) indolent lesions persist, (iii) extension of the lesion with progressive destruction of tissues, (iv) bronchial wall erosion with partial or complete occlusion of bronchial lumen with establishment of localised obstructive emphysema or atelectasis and at times with distribution of tubercle bacilli to other parts of the lung and establishment of number of new lesions, (v) erosion of blood vessels with wide spread

distribution of tubercle bacilli (miliary tuberculosis) or with establishment of localised lesions at distant sites, (vi) subsequent reactivity of the lesion or (vii) re-infection, endogenous or exogenous.

One year after infection, the primary focus in the lung may extend to involve the pleural sac and cause an effusion. Biological healing of the primary foci takes a very long time. The bacilli may persist in calcified lesions for many years and perhaps biological healing is never complete in some cases. Anatomically, the calcified foci shrink and show regressive changes in which they first become denser and then by eventful absorption of calcium salts, the density may in turn decrease, finally, after some decades they may disappear. Ninety percent of all cases of tuberculous meningitis and miliary tuberculosis arise in the first few weeks or months after primary infection. Most of bone and joint lesions appear within two to three years of primary infection. Hematogenous skin lesions, papulonecrotic tuberculides appear as a rule, within two years, but isolated lesions of lupus vulgaris or verrucose tuberculides appear many years afterwards. Tuberculosis of the genital tract is uncommon in either sex before puberty.

Common Tuberculous Intrathoracic Lesions :

1. Segmental Lesion :

On occasion the initial lesion in the lung is not confined to a small focal area, but extends into the surrounding tissue or segments. Such lesions may involve several lobules or most of lobe. Though there may be symptoms; not infrequently, extensive pulmonary lesions are detected roentgenographically, in children, who have no complaints and had no physical finding. Average age incidence is 5 to 6 years. The lesions are frequently seen in right upper and middle zones.

A hilar lymphode involvement is an almost constant feature of pulmonary tuberculosis in childhood. The infection of the lymphnode undergoes similar changes as that of paranchymal lesion until calcification is complete it has the same dangerous of local extension and hematogenous spread.

There may be intra-luminal extension of the tuberculous process usually in a lymphnode, through the bronchial wall with formation of an ulcerative or granulomatous lesion. This may partly or completely obstruct the luman of the broncheus which ultimately would lead to the dissemination of the infection material to the other portions of tracheo-bronchial tree with



establishment of non-caseating broncho-pneumonia. Extraluminal occlusion may be partial or complete, brought about by enlarged, adjacent tuberculous lymphnodes without erosion through the bronchial wall. Partial compression gives rise to emphysema of the segment but in complete obstruction absorption atelectasis occurs. In each instance there may be a tuberculous pneumonitis in all or part of this involved pulmonary area.

Occasionally children with bronchial erosion do not develop either bronchial obstruction or a radiological segmental lesion. Instead, small areas of broncho-pneumonic changes appear diffusely throughout the lung field, these without the chemotherapy would ultimately coalesce to form extensive caseating broncho-pneumonia. It may be localised in one area of the lung or it may be widely disseminated children with this lesion tend to be quite sick.

## 2. Pleurisy :

Though the pleura is often involved it is less frequently found on clinical or radiological examination. It may occur as a dry fibrinous, pleurisy, as a serous effusion and rarely as a necrotic involvement of pleura, stemming from a contagious caseous focus in the lung.



Most effusions occur during the first few months after primary infection but some times clear evidence, such as glandular calcification may exist to show that the infection has been present since long. It does not occur below the age of 18 months, and rarely below five years. The common age incidence is between 10-12 years. Most authors have found that pleural effusion, like, primary complex itself, is more common on right side, and before the days of chemotherapy about 5% of children developed effusion on the other side also, within a year and occasionally within a few weeks after first infections. All pleural effusions should be regarded tuberculous until proved otherwise. There is a disagreement regarding the route by which the tubercle bacilli reach the pleura. According to Thompson and Land, the bacilli reach the pleura from an active primary lesion of the lung. Pleural effusion arises on the same side as the primary lesion in the lung. It is rarely seen on the contra-lateral side. Sibly believes that it is due to a haematogenous spread as it is associated with miliary tuberculosis and other extra-pulmonary complications. The diagnosis depends on the tuberculin test, chemistry and cytology of pleural fluid and demonstration of organisms in pleural fluid. Pleural effusion may get complicated with bilateral effusion, meningitis, pericarditis and skeletal tuberculosis.

### 3. Miliary Tuberculosis :

It is a blood borne infection characterised by multiple tubercle formations. Tubercle bacilli become lodged in the small capillaries, a lesion develops at each site and necrosis tends to develop rapidly in each of small foci. The symptoms are usually those of the general infection, initially there may be no physical signs. If choroidal tubercles are visible the diagnosis can be made before the results of tuberculin test X-ray are known. The distribution of the lesions may be limited to the lungs or may include other viscera i.e. liver, kidney, spleen and brain.

In miliary tuberculosis three major types of radiological picture are seen and in any of them other radiological evidence of tuberculosis may be present, such as a primary complex, mediastinal lymphadenitis, segmental lesion or pleural effusion.

1) The "snowstorm" type : In which innumerable small nodules are scattered equally throughout all the areas of both lungs. They are best seen in the diamond shaped spaces between the ribs; round, or approximately so, they vary in size from a millimetre upwards and when large enough have a centre denser than the edge which tends to be indistinct.

ii) The "hard" chronic type : These lesions are fewer in number and usually larger and give the impression of a lighter and more intermittent spread than that which produces the snowstorm film - the so-called sub-acute or chronic types of miliary lesions fall into this category.

iii) The "mixed" type : Usually found in infants or young toddlers who are ill with extensive disease, some of which is bronchogenic aerogenous spread and may contain areas of cavitating disease but in addition in the less affected areas, evidence of miliary spread may also be present.

There were well authenticated examples of survival after miliary tuberculosis before the introduction of chemotherapy but they were rare, the prognosis was bad for most cases developed fatal meningitis.

#### 4. Tuberculous Empyema :

Generally speaking no child treated with chemotherapy at the onset of a pleural effusion develops an empyema. Miller, Seal and Taylor (1963) suggested that many effusion can become purulent, if sufficient tubercle bacilli are present, if the original cause of the effusion has been, rupture of a caseous lymphnode and occasionally, if there is a double infection with

*Mycobacterium tuberculosis* and pyogenic organisms. Price, on the other hand, says that serious effusions do not become purulent and that empyemata do not occur in children except as a complication of pneumothorax or when haematogenous foci form in the pleura without underlying pulmonary involvement.

The possibility of empyema should be suspected whenever a pleural effusion becomes encysted or takes longer than usual to absorb. The general physical condition of the child is no indication of the character of the encysted effusion and diagnostic aspiration is required. If the empyema is small than aspiration and chemotherapy with careful supervision and adequate physiotherapy are sufficient. With larger collections or if the pleuras have become thickened so that the affected lung is immobilized, the possibility of surgical treatment will arise.

#### Extra Thoracic Tuberculous Lesions :

##### 1. Glandular Lesions :

In cases of extra-thoracic lesions, the cervical, preauricular, mesenteric or original lymphnodes are enlarged. Such enlargements are seen in older age group after five years of age.



Infection of the cervical lymphnodes is, in most instances, secondary to tuberculous tonsils or to a pulmonary lesion. Nodes of the both sides are affected frequently, initially discrete, firm and freely mobile, later when they become caseous, there is tendency to erosion of the capsule, matting together with the adjacent nodes forming an irregular mass. The mass becomes attached to other adjacent structures. Subsequently the caseous mass may liquify and not infrequently the overlying skin is perforated forming a sinus. Retropharyngeal lymphnode involvement may give rise to osseous lesion in the cervical vertebrae, and retropharyngeal abscess. In spite of this, most of the nodes undergo resolution and calcification before extensive caseation occurs. Other glandular involvement may occur in axilla, groin and occipital region, usually occurring secondary to tuberculosis of skin, but are of rarer occurrence.

## 2. Abdominal Tuberculosis :

Primary abdominal infection may occur alone or simultaneously with primary infection elsewhere, usually in the oropharynx or the lungs. When it does occur the usual sequence of events ensues and a small lesion in the bowel wall is associated with enlargement and caseation in the regional mesenteric nodes. Localised clinical illness



at this stage is almost always due to complications of the mesenteric adenitis, which are as follows :-

- a) A node may rupture and the liberation of caseous material and possibly tubercle bacilli into the peritoneal cavity produces a reaction analogous to that of a pleural effusion.
- b) The node may soften and slowly involve neighbouring coils of bowel in a plastic peritonitis causing acute or sub - acute intestinal obstruction.
- c) A single loop of bowel may become adherent over the surface of a node and given rise to acute intestinal obstruction.
- d) As in peripheral adenitis, healing mesenteric nodes may become active again after non-specific infection.

Mild constitutional and vague general symptoms are the main features of this disease. The physical findings are rarely supporting. It is suspected on the findings of tuberculin conversion and raised E.S.R. in the absence of active pulmonary disease. The diagnosis is supported by the changes observed during the therapy (Gefel et al, 1963) and is proved after leprotony. It has been classified in the following types :-

- i) Intestinal type : It has two forms; ulcerative and hypertrophic, the latter is more common in cases of primary infections.
- ii) Glancular type or mesentric adenitis.
- iii) Peritoneal type : It has got 3 sub-types; ascitic-most common, plastic or fibrinous and miliary (rare).
- iv) Combined type.

### 3. Primary Tonsillar Infecction :

In tonsils, the primary focus is localised in the crypts. It is small and not as a rule demonstrable by examination in vivo, and the tonsil need not be enlarged (Wallgren, 1948). Observations of Miller et al (1963) have shown that unequal tonsillar enlargement associated with regional adenitis should always arouse suspicion of a primary tuberculous infection, and this unequal enlargement was striking in ten of their twelve children. The inequality was noticeable even if both tonsils were large. After removal of the tonsil the tuberculous lesions are easy to detect by histological examination.

### 4. Tuberculosis of Skin :

Ghon (1912) in his monograph on primary lung infection recognised that primary complex could occur on skin or a mucosal surface. Since then many case reports

of primary skin infection have appeared but almost always have been limited to one or two cases.. Tubercle bacilli invade the skin or mucus membrane through abraisions; the common sites are the lip, nose, chin, extremities and genital region. There is an accompanying involvement of regional lymphnodes to complete the primary complex. Its common forms met within childhood are lupus vulgaris, sacrofuloderma, lichen sacrofulosus and tuberculides.

#### 5. Congenital Tuberculosis :

The cases of congenital tuberculosis reported upto 1945 have been critically analysed by Hughesdon (1946), who added a few new cases. Infants with truly congenital tuberculosis may give no indication of illness before their sudden death. Attention should be called to the infant by the development of the nasal discharge, cough, dyspnoea, lethargy, anorexia, failure to gain weight or by the passage of bloody stool indicating intestinal ulceration. By the time the disease is suspected, lungs ordinarily show advanced, wide-spread areas of consolidation and hilar gland enlargement, with or without cavitation and miliary spread. The course is long and even in the face of vigorous therapy progression is the rule.



### Allergic Manifestations in Primary Tuberculosis :

These manifestations are owing to the allergic reaction to the tuberculo-protein circulating in the body of the individual. Such manifestations are as under :-

i) Phlyctenular Conjunctivitis : It is characterised by a lesion seen as a small, grey spot at the limbus in one or both eyes; a single spot with a leash of conjunctival vessels running towards phlyctenules.

Phlyctenules are accumulations of lymphoid cells beneath the epithelium of cornea or conjunctiva. This conjunctivitis is seen most often between 5 and 15 years.

ii) Erythema Nodosum : These are believed to result from hyper-sensitivity to tuberculo-protein in cases of tuberculosis. These lesions are characterised by the development of tender, painful indurated, shining, elevated ovoid patches 1 to 3 cms. in diameter, usually symmetrically distributed over the shins, calves, knees, buttocks and occasionally the arms. The indurations decrease after 1-2 weeks. Crops of lesion occur, generally over a period of 3-6 weeks, later recurrences are unusual.

iii) Allergic Lymphadenitis : The adenitis appears rather suddenly in association with constitutional symptoms. In case of cervical adenitis it is bilateral. The gland recedes in two to three weeks' time and with it the constitutional symptoms also disappear.

iv) Allergic Pleural Effusion : It appears at the time of tuberculin conversion and is similar to other form described already.

Diagnosis of Primary Tuberculosis :

Early diagnosis of primary tuberculous infections is of paramount importance in order to reduce the incidence and prevent the complications of the disease. Besides the positive history of contact, haematological examination, E.S.R. and bacteriological investigations, a positive tuberculin and abnormal roentgenographic appearance are the essential criteria for the absolute confirmation of the diagnosis. Lesions are termed inactive when constitutional disturbances, physical signs, radiologic evidence, bacteriologic examination and other investigations are negative (Sekulick, 1955).

Tuberculin Test :

The tuberculin test was discovered by Von Pirquet in 1907 and was established as a principal case-finding agent. Various methods have been employed from time to time by different workers; viz. Von Pirquet Test (scarification of the skin through Old Tuberculin), the Mantoux Test (intradermal injection of P.P.D.), percutaneous Tests (Vollmer patch test, the jelly test), Heaf's Multiple puncture Test and Tine Test. No other method or no other



material has been as extensively used in this country or perhaps in the whole world as the Mantoux technique using P.P.D.

Mantoux Test :

Charles Mantoux suggested a dose of 1 T.U. (1/20th c.c. of a 1.5000 dilution of O.T.). Tuberculin used for this test is of 2 types - (i) Old tuberculin, prepared by growing tubercle bacilli on artificial mediums, then entire culture products is filtered and the filtrate is concentrated and used, (ii) Purified protein Derivative (P.P.D.).

Around 1934, Seibert and Munday having realised that the anti-genicity of tuberculin was related to its molecular size, produced the substance known as purified protein derivative, in which the proteins were of a relatively small molecular size, this was a major advance. In 1941, the W.H.O. established P.P.D.-S at international standard for Mammalian tuberculosis. In 1958, however, it was found that a new tuberculin P.P.D. R.T. 23 in a solution containing Tween 80 was more potent than P.P.D.-S. P.P.D. is obtained by growing tubercle bacilli of strain RT 23 on protein free culture medium, from which protein (Tubercle bacillus protein) is then precipitated. P.P.D. is thus more purified product than is old tuberculin and

it has been adequately demonstrated that it is an effective skin testing material, and after dilution can be stored in refrigerator for 6 months and marketed in dry state, which is diluted at the time of use.

Dose of P.P.D. :

The potency of tuberculin (P.P.D.) may be expressed in the terms of international tuberculin unit called T.U. One T.U. is equivalent to .00002 mg. of P.P.D., i.e. 1:50,000 or 0.01 mg. O.T. There is a great controversy regarding dosage of P.P.D. to be employed. Some authors have suggested 10 T.U. for routine check-up, while others suggest 1 T.U. because with 10 T.U. there are chances of severe reaction and false positive results. Agarwal (1962) and Eagle (1962) have advocated 1 T.U. of PPD RT 23 with tween 80, as the ideal testing dose for our country. The standardization of tuberculin reaction, as described by W.H.O. (1959) is as follows :

- a) Induration of 10 mm. are more regarded as positive (but drugs and diseases should be kept in mind).
- b) Induration between 5-10 mm. is doubtful and in these cases test should be repeated.
- c) Induration below 5 mm. is negative.

Agarwal and Wagle (1962), in their separate studies have recommended an induration 8 mm. and above to consider the test as positive.

#### Hematological Examination :

Haemoglobin percentage, total and differential leucocytic counts, agglutination, complement fixation and the haemagglutination tests do not serve as a diagnostic or prognostic purpose.

#### Sedimentation Rate :

Wintrobe has noted that raised sedimentation rate in the presence of the other evidences for the etiologic diagnosis, suggest acute disease process. The rate varies with the extent and nature of this disease. He further emphasized that it is a guide to the process of infection, particularly in tuberculosis. Though a normal sedimentation rate does not necessarily mean that all is well, occasionally specially in cachaxia. The importance of sedimentation rate lies more in judging the prognosis and course of the disease process.

#### X-Ray Examination :

"In the diagnosis of pulmonary tuberculosis at any rate Roentgen tube has superseded Laennec's tube", Burton Wood (1930). In fact, it is the only method as has been

shown by results of mass radiography in detection of the cases. The primary focus is seldom shown in recent primary pulmonary tuberculosis, the X-ray changes are usually those of hilar infiltration, which owing to its smallness and its position is obscured by the air content of the lung. The hilar changes consist of enlargement and increased density of the root shadow with streaky or cloudy diffuse limitation. In lateral (oblique) view the retrocardial space is occupied by the shadow of enlarged glands. If the primary focus is visible, it presents itself as a homogeneous, cloudy, rather diffuse shadow, often seemingly in direct connection with the enlarged hilar shadow.

There is nothing characteristic of the X-ray picture of primary tuberculosis. The same changes may encountered in several other disease for example atypical pneumonia, common pneumonic lesions etc. The X-ray changes in cases of pulmonary primary tuberculosis remain for atleast 3-4 months without any sign of decreasing and the hilar shadow become gradually smaller, more distinctly limited and more dense (Wellgren and Wegelius, 1949). After 2-3 years calcification is seen in the necrotic parts of the hilar glands and in the primary focus. If not previously seen in the X-ray film, the primary focus become obvious after calcifications.



Vasantkumar et al (1976) studied 1500 cases of clinically suspected primary complex radiologically. A dynamic radiological classification of primary complex was attempted. The pathological significance of interlobar pleural opacification had been stressed. The various features observed on the chest roentgenogram were numbered and tabulated as :-

1. Primary focus with parifocal inflammation.
2. Lymphangitis.
3. Hilar adenitis.
4. Mediastinal adenitis.
5. Interlobar pleural opacification.
6. Primary focal calcification.
7. Calcification of Lymphatics -
  - a) Hilar.
  - b) Mediastinal.
8. Glandular calcification.
9. Pleural effusion.
10. Pleural thickening.
11. Pleural calcification.
12. Atelectasis.
13. Bronchiectasis reversible, irreversible.
14. Miliary tuberculosis.
15. Bronchopneumonia.
16. Associated caries of thoracic spine.



17. Tubercular pneumonia.
18. Obstructive emphysema.
19. Extrapulmonary calcification.
20. Cavitating lesion.

In his study, it was noted that 76% of cases and 63% cases of Mantoux positive, the minor interlobar pleural fissure was opacified. So this work revealed that the opacification of interlobar pleural fissure in the chest skiagram of the children were of significant value in diagnosis of primary complex.

Ramchandran and Mukundan (1976) reported to approach radiological shadows in tuberculosis 1. How long after proper treatment, do lymphnodes and / or parenchymal lesions get calcified ? 2. How long after treatment do the intrathoracic lymphnode shadow diminish in size of disappear completely ?

How often does intracranial calcification occur in children suffering from tuberculous meningitis ? Is there a discernible relationship between intrathoracic and intracranial calcification ? An attempt was made to answer above questions. The X-ray analysis of the report taken from the children attending T.B. Clinic, in the department of Paediatric, Medical College, Tanjavur. The children were registered as per criteria of Ramchandran

(1971), namely - suspicious symptoms, mantoux positive, radiological evidence of Tuberculosis and chest X-ray.

Out of 3000 children registered, 630 X-rays only were available for studies.

In the radiological study of 1630 cases, 527 had only lymphnode involvement, 223 had node with parenchymal focus and 86 had multiple infiltration. So lymphnode constitute above 50% of radiological findings.

Payre quoted Miller (1963) reported that 32.7% of children with pulmonary primary infection had radiological evidence as primary complex. In majority of X-ray the infiltration is in the right upper lobe and the lymphnode is often superior mediastinal and sometimes right hilar. In cases with right lower lobe infiltration, the lymphnode are often right interior and posterior bronchial.

#### Lymphnode enlargement

| Location                      | No. of cases |
|-------------------------------|--------------|
| Superior mediastinal adenites | 215          |
| Right hilar adenites          | 165          |
| Left hilar adenites           | 36           |
| Bilateral hilar adenites      | 53           |
| All glands                    | 58           |

## Lymphnode with parenchymal infiltration

| Location                      | No. of cases |
|-------------------------------|--------------|
| Superior mediastinal adenites | 72           |
| Right hilar adenites          | 96           |
| Left hilar adenites           | 32           |
| Both right and left adenites  | 23           |

## Progressive primary complex

| Location                             | No. of cases |
|--------------------------------------|--------------|
| Lymphnode with multiple infiltration | 86           |
| Consolidation                        | 134          |
| Effusion                             | 23           |
| Broncho-pneumonia                    | 74           |
| Miliary                              | 74           |

In this series 86 cases had multiple infiltration distribution all over the lung fields and in 60% X-rays, they were at right side.

### Pleural effusion :

Twenty three cases reported with pleural effusion (Miller, 1963) reported more right sided effusions, almost all pleural effusions are post primary. On X-ray examination more often a horizontal line is seen in children due to fluid in inter-lober septum or to a co-existing segmental lesion. Quite often the pleural effusion is revealed by vertical line following the attachment of parital pleura to appex of the lung.

The radiologicalevidence of pleural effusion disappears within 4 weeks on sterioid therapy. Miller (1963) reported the clearance of effusion radiologically depends on the duration of effusion prior to therapy.

### Miliary :

Miller (1963) miliary tuberculosis can be of 3 types in radiological appearance. Snowstorm, hard chronic type, mixed type.

Lincoln's (1963) criteria divides radiological appearance into classical and sub-miliary. The sub-miliary shadow become evident when film is looked at slanting position. X-ray give impression of granularity. Twenty out of 74 shows miliary lesions, rest associated with either superior mediastinal nodes, hilar adenopathy or parenchymal patches.



Consolidation - 123 cases shows consolidation out of which 99 cases were right sided similar to infiltration, 20% of these cases shows AFB in gastric leavage.

Calcification - Calcification of tuberculosis lymphnodes and parenchymal lesion is end resulting the disease of all above cases.

According to Payne quoted by Miller (1963) calcification occurs after 4 years of treatments and 80% calcified after 3 years of treatments.

Tubercular meningitis - 55 skull X-ray children with tubercular meningitis were taken. Only 6 shows calification after 1 1/2 years of treatment. The incidence of intracranial calcification was very low compared to Corber's figure of 48.4% and Miller's (1963) 37.5%. This was attributed due to the use of corticosteroid therapy and poor nutritional status of children.

As study carried out by S. Malik, K.L.Narasimharao (Nov., 1983).

A child with clinical and radiological features of solitary mass lesion in the lung proved to be tuberculoma was reported for its rarity.

Tuberculoma is a localised granulomatous lesion and is manifestation of primary tubercular infection. Large tuberculoma is rare in children and is extremely rare in children under 2 years. Tuberculoma when associated with mediastinal gland enlargement usually have anterior group of glands involved.

A study carried out by N.R. Bhandari and associates, Indian Paediatric J. ( July, 1984).

B.C.G. test was positive in 90.9% cases of tuberculosis as compared to tuberculin test which was positive to 47.2%. The effect of malnutrition on the diagnostic sensitivity of BCG test is not significant local complication was observed in 1.2% cases. BCG test is safe and simple diagnostic method for diagnosis of childhood tuberculosis and at the same time provides immunity to those who need it.

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## MATERIAL AND METHODS

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The present study was carried out in the department of Radiology, M.L.B. Medical College, Jhansi (U.P.). Children upto 12 years of age who were either admitted for treatment in children wards or attended O.P.D. of Paediatrics and Well Baby Clinic were taken into account.

The positive cases of primary tuberculosis were taken in our study. Their diagnosis were based on detailed history, clinical examination, laboratory examination and radiological examination.

### History of Contact :

Patient with positive history of contact from parents or from relative and from neighbours were taken into account.

The nutritional status of individual patients were studied as per Indian Academy of Paediatrics Classification (1972) of Malnutrition.

Normal 80% by weight of the 50th percentile of Harward Standard.

|       |     |    |   |     |        |
|-------|-----|----|---|-----|--------|
| Grade | I   | 71 | - | 80% | - do - |
| Grade | II  | 61 | - | 70% | - do - |
| Grade | III | 51 | - | 60% | - do - |
| Grade | IV  | <  | - | 50% | - do - |



Besides this, cases were also studied as per the age group :-

|                  |   |                            |
|------------------|---|----------------------------|
| New born         | - | First 4 weeks after birth. |
| Infants          | - | First year.                |
| Toddler          | - | 1-3 years.                 |
| Pre-school child | - | 3-6 years.                 |
| School age child | - | 6-10 years.                |
| Adolescent       | - | 10-12 years.               |

Besides recording name, age, sex, address, socio-economic status, occupation of parents, birth order or child in the family and per capita income, following facts were recorded on each cases on specially designed proforma.

From parents and other family members, detailed history was obtained regarding present illness in chronological order.

#### PAST ILLNESS :

A detailed past history of pertusis, measles, malaria, worm infestation etc. were noted.

#### FAMILY HISTORY :

An enquiry was made about a definite history of tuberculosis in parents, siblings, near relatives,

neighbours and other care takers. For example, chronic cough, haemoptysis, prolong fever, dyspnoea, weight loss, in suspected cases of family members were sent to T.B. Clinics for diagnosis and treatment of tuberculosis.

CLINICAL EXAMINATION :

A thorough clinical examination was done including general appearance, pallor, anaemia, cyanosis, clubbing, oedema, fascial look, hair changes, skin condition, body weight, various vitamin deficiency state.

- Presence or absence of fever, cough, excessive sweating.
- Headache, vomiting, convulsions.
- Loss of weight, change in behaviour.
- Loss of appetite.
- Diarrhoea, constipation, pain in abdomen.
- Lymphadenopathy - cervical, axillary, inguinal or abdominal.
- Ascitis.
- Hepatosplenomegaly.
- Pulmonary findings.
- Sign of central nervous system involvement.

INVESTIGATION :

The children of study group were subjected to some or all of the following laboratory investigations:-

Blood

- (a) Haemoglobin estimation (Sahli's Haemoglobino-meter).
- (b) Total and differential white blood cell count (Thomazeiss instrument).
- (c) Estimation of Erythrocyte sedimentation rate (Wintrobe's method).

X-ray chest

Routine examination of chest, P.A. Projection or A.P. Projection were done. In certain instance, lateral decubitus and laredotic projection view were also taken.

Mantoux test

Inject 0.1 ml. of PPD taking all aseptic precautions. The volar aspect of right fore-arm was chosen as site for Mantoux test.

The test was read after 72 hours of injection. Transverse and vertical diameter were measured.

Induration 7 10 mm. after 72 hours was considered as Mantoux test positive.

The basic materials used in our study are :-

1. Children attending Paediatric O.P.D. and indoor upto age of 12 years.
2. X-ray machines and accessories.
3. X-ray films and cassettes.
4. Fixer and developer.
5. Viewing boxes.
6. Tuberculin test (Mantoux).

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**OBSERVATIONS**

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|       |       |
|-------|-------|
| 10.30 | 10.30 |
| 11.30 | 11.30 |
| 12.30 | 12.30 |
| 13.30 | 13.30 |
| 14.30 | 14.30 |
| 15.30 | 15.30 |

## OBSERVATION

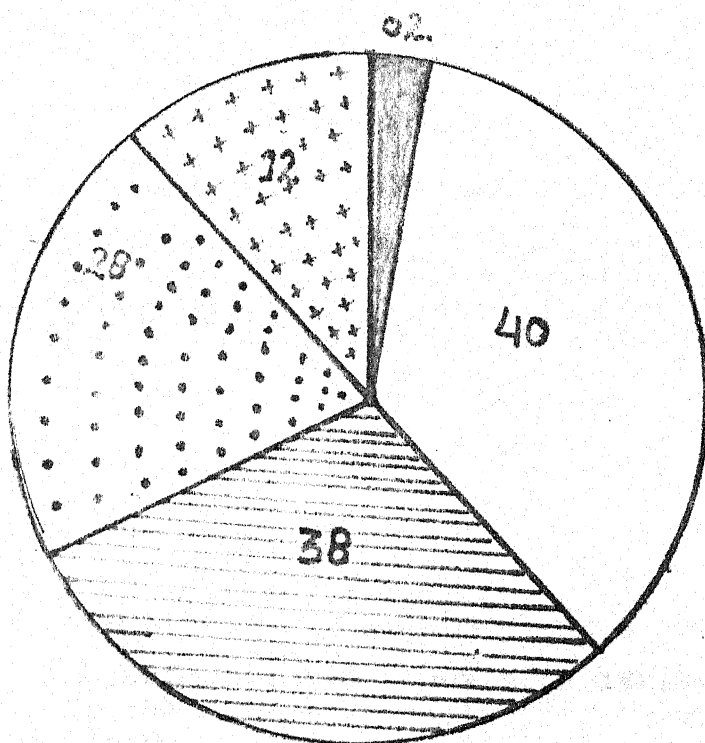
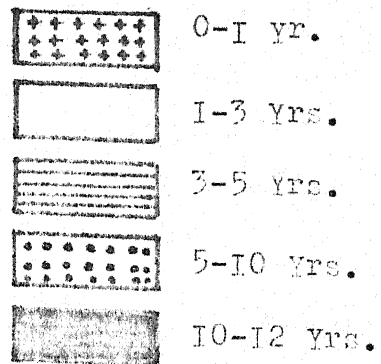
The present study "Radiological evaluation of primary tuberculosis in mantoux test positive children" attending Out Patient Department of M.L.B. Medical College, Jhansi, is conducted. Total number of 120 children attended the outdoor and indoor department of Pediatrics were included. The children ranging from the age of 0-12 years, having relevant symptoms and signs suggestive of primary tuberculosis such as - Pyrexia, Pallor, Cervical adenitis, Failure to thrive, Cough with or without expectoration, recurrent respiratory tract infection, breathlessness, gastro-intestinal symptoms etc.

Out of total 120 cases 0-1 year of age were 12 cases (10.00%), 1-3 years 40 (33.34%), 3-5 years 38 (31.67%), 5-10 years 28 cases (23.33%) and 10-12 years 2 cases (1.67%).

TABLE - I

Showing incidence of primary tuberculosis with relation to age.

| Age          | No. of cases | Percentage |
|--------------|--------------|------------|
| 0 - 1 yr.    | 12           | 10.00      |
| 1 - 3 yrs.   | 40           | 33.33      |
| 3.- 5 yrs    | 38           | 31.67      |
| 5 - 10 yrs.  | 28           | 23.33      |
| 10 - 12 yrs. | 02           | 01.67      |



PERCENTAGE OF PRIMARY COMPLEX IN RELATION TO AGE

Figure I.

Out of total 120 cases, males were 77 cases (64.16%) and females were 43 cases (35.84%).

TABLE - II

Showing sex-wise distribution of the cases

| Age in yrs. | Male |       | Female |       |
|-------------|------|-------|--------|-------|
|             | No.  | %     | No.    | %     |
| 0 - 12      | 77   | 64.16 | 43     | 35.84 |

Out of 77 male 0-1 year age group were 6 cases (5.0%), 1-3 years 34 cases (28.34%), 3-5 yrs. 24 cases (20.00%), 5-10 yrs. 11 cases (9.17%), 10-12 yrs. 2 cases (1.67%).

Out of 43 female, 0-1 year age group were 6 cases (5.0%), 1-3 yrs. 6 cases (5.0%), 3-5 yrs. 14 cases (11.67%), 5-10 yrs. 17 cases (14.17%).

TABLE - III

Showing age and sex-wise distribution of cases

| Age group in yrs. | Male |       | Female |       |
|-------------------|------|-------|--------|-------|
|                   | No.  | %     | No.    | %     |
| 0 - 1             | 06   | 5.00  | 06     | 5.00  |
| 1 - 3             | 34   | 28.33 | 06     | 5.00  |
| 3 - 5             | 24   | 20.00 | 14     | 11.66 |
| 5 - 10            | 11   | 9.17  | 17     | 14.17 |
| 10 - 12           | 02   | 1.67  | -      | -     |



Total no. of cases with history of contacts were 60. Out of 60, history of contacts from parents and sibs were 28 cases (23.33%) and from other relation & neighbourhoods were 32 cases (26.67%).

TABLE - IV

Correlation of proved tubercular contact with occurrence of primary tuberculosis.

| History                              | No. of cases | Percentage |
|--------------------------------------|--------------|------------|
| From parents & sibs                  | 28           | 23.33      |
| From other relation & neighbourhoods | 32           | 26.67      |

Out of 120 cases Past history collected from individual cases were - measles 20 cases (16.67%), Whooping cough 4 cases (3.33%), repeated respiratory tract infections 28 cases (22.33%), gastro-intestinal 26 cases (21.67%) and mumps 2 cases (1.67%).

TABLE - V

Showing correlation of relevant past illnesses with occurrence of primary tuberculosis.

| Past illness                | No. of cases | Percentage |
|-----------------------------|--------------|------------|
| Measles                     | 20           | 16.67      |
| Whooping cough              | 04           | 03.33      |
| Respiratory tract infection | 28           | 23.33      |
| Gastro-enteritis            | 20           | 21.67      |
| Mumps                       | 02           | 01.67      |

Out of 120 cases, clinical symptom & sign collected as Pyrexia 61 cases (50.83%), Pallor 40 cases (33.33%), Cough with or without expectoration 70 cases (58.33%), Failure to thrive 44 cases (36.67%), Breathlessness 27 cases (22.50%), Recurrent respiratory infections 44 cases (36.67%), Cervical adenitis 54 cases (45%), Generalised adenitis 20 cases (16.66%), Hemoptasis 4 cases (3.33%), GIT symptoms 31 cases (25.83%).

TABLE - VI

Showing clinical signs and symptoms

| Symptoms & signs                      | No. of cases | Percentage |
|---------------------------------------|--------------|------------|
| Pyrexia                               | 61           | 50.83      |
| Pallor                                | 40           | 33.33      |
| Cough with or without expectoration   | 70           | 58.33      |
| Failure to thrive                     | 44           | 36.67      |
| Breathlessness                        | 27           | 22.50      |
| Recurrent respiratory tract infection | 44           | 36.67      |
| Cervical adenitis                     | 54           | 45.00      |
| Generalised adenitis                  | 20           | 16.66      |
| Haemoptysis                           | 04           | 03.33      |
| G.I.T. symptoms                       | 31           | 25.83      |

Out of 120 cases radiological presentation was Hilar adenitis 88 cases (73.33%), Paratracheal adenitis 50 cases (41.67%), Lymphangitis 85 cases (70.83%), Mottling 65 cases (54.16%), Consolidation 14 cases (11.67%), Collapse 8 cases (6.67%), Pleural effusion 8 cases (6.67%), Cavitation 6 cases (5.0%), Pleural thickening 4 cases (3.33%).

TABLE - VII

Showing radiological presentation of primary tuberculosis

| Radiological appearance | No. | Percentage |
|-------------------------|-----|------------|
| Hilar adenitis          | 88  | 73.33      |
| Paratracheal adenitis   | 50  | 41.67      |
| Lymphangitis            | 85  | 70.83      |
| Mottling                | 65  | 54.16      |
| Consolidation           | 14  | 11.67      |
| Collapse                | 08  | 06.67      |
| Pleural effusion        | 08  | 06.67      |
| Cavitation              | 06  | 05.00      |
| Pleural thickening      | 04  | 03.33      |

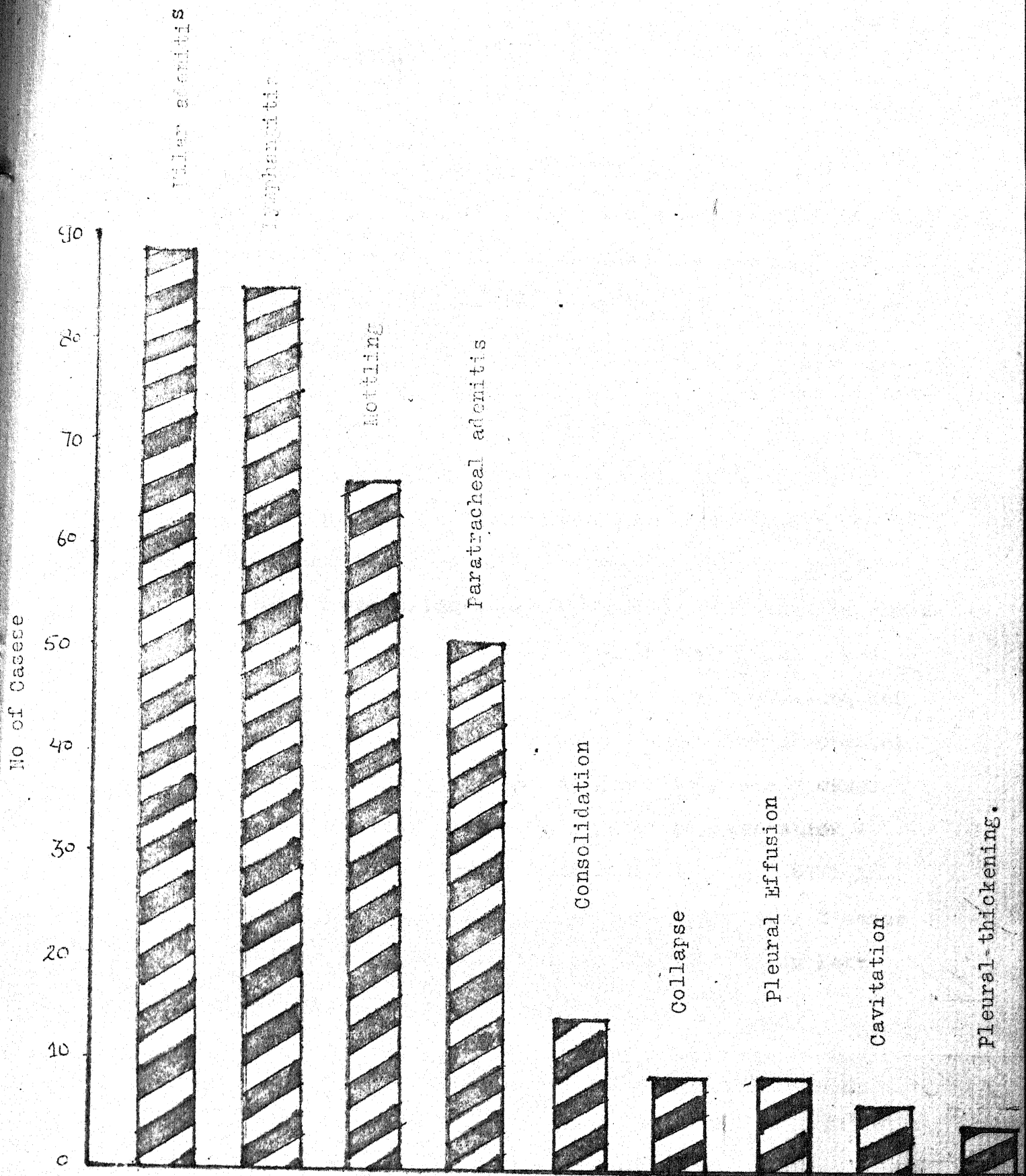


Figure 2.

Radiological Presentation of Primary Tuberculosis



Out of 88 cases of hilar adenitis 41 cases (34.17%) in right side, 22 cases (18.33%) in left side and bilateral 25 cases (20.83%). In 50 cases of paratracheal adenitis 26 cases (21.67%) right side, 13 cases (10.83%) left side and bilateral 11 cases (9.17%). In 85 cases of lymphangitis 38 cases (31.67%) in right lung, 21 cases (17.5%) in left lung and bilateral 26 (21.67%). In 65 cases of mottling 37 cases (30.83%) in right lung, 14 cases (11.67%) in left lung and bilateral 14 cases (11.67%). In 14 cases of consolidation, 10 cases (8.33%) were in right lung; and 4 cases (3.33%) were in left lung. In 8 cases of collapse, 4 cases (3.33%) in right lung and 4 cases (3.33%) in left lung. In 8 cases of pleural effusion 4 cases (3.33%) on right side and 4 cases (3.33%) on left side. In 6 cases of cavitation 4 cases (3.33%) in right lung and 2 cases (1.67%) in left lung. In 4 cases of pleural thickening 2 cases (1.67%) in right lung and 2 cases (1.67%) in left lung.

**TABLE - VIII**

**Showing Lung with distribution of Radiological features  
in Primary Tuberculosis.**

| <b>Radiological appearance</b> | <b>RT</b> | <b>LT</b> | <b>BL</b> |
|--------------------------------|-----------|-----------|-----------|
| Hilar adenitis                 | 41        | 22        | 25        |
| Parancheal adenitis            | 26        | 13        | 11        |
| Lymphangitis                   | 38        | 21        | 26        |
| Mottling                       | 37        | 14        | 14        |
| Consolidation                  | 10        | 4         | -         |
| Collapse                       | 4         | 4         | -         |
| Pleural effusion               | 4         | 4         | -         |
| Cavitation                     | 4         | 2         | -         |
| Pleural thickening             | 2         | 2         | -         |

Out of 120 cases, the distribution of primary complex were typical primary complex 53 cases (44.17%) and Hilar adenitis alone 35 cases (29.16%).

TABLE - IX

Showing the distribution of primary complex

| Lesion                  | No. of cases | Percentage |
|-------------------------|--------------|------------|
| Typical primary complex | 53           | 44.17      |
| Hilar adenitis (alone)  | 35           | 29.16      |

Out of 120 cases, the distribution of the disease zone wise are typical primary complex, Right lung 31 cases (25.83%) left lung 8 cases (6.67%) and bilateral 14 cases (11.67%). Hilar adenitis right lung 20 cases (16.67%), left lung 5 cases (4.17%) and bilateral 10 cases (8.33%).

TABLE - X

Showing types &amp; distribution of primary complex

| Lesions                 | <u>Total cases</u> |       | <u>Right lung</u> |       | <u>Left lung</u> |      | <u>Bilateral</u> |       |
|-------------------------|--------------------|-------|-------------------|-------|------------------|------|------------------|-------|
|                         | No.                | %     | No.               | %     | No.              | %    | No.              | %     |
| Typical Primary Complex | 53                 | 44.17 | 31                | 25.83 | 8                | 6.67 | 14               | 11.67 |
| Hilar adenitis alone    | 35                 | 29.16 | 20                | 16.67 | 5                | 4.17 | 10               | 8.33  |

Out of 120 cases, the distribution of primary complex were Right lung 38 cases, Left lung 15 cases. The zonal distribution of cases were Right upper zone 20 cases. Right middle zone 16 cases and Right lower zone 2 cases. Left lung 15 cases, Left upper zone 8 cases, Left middle zone 5 cases and Left lower zone 2 cases.

TABLE - XI

Showing zone-wise distribution of primary complex in lung.

| Zone-wise   | <u>Right lung</u> |       | <u>Left lung</u> |      |
|-------------|-------------------|-------|------------------|------|
|             | No.               | %     | No.              | %    |
| Upper zone  | 20                | 16.67 | 08               | 6.67 |
| Middle zone | 16                | 13.33 | 05               | 4.16 |
| Lower zone  | 02                | 01.67 | 02               | 1.67 |

Out of 120 cases, the clinico-radiological survey was done.

Pyrexia 61 (50.83%), radiological features were hilar adenitis 40 cases (33.33%), Paratracheal adenitis 26 cases (21.67%), lymphangitis 45 cases (37.5%), Mottling 35 cases (29.16%), consolidation 4 cases (3.33%), collapse 4 cases (3.33%), Pleural effusion 2 cases (1.67%), Pleural thickening 1 case (0.83%),



cavitation 4 cases (3.33%), Pallor 40 cases, (33.33%), radiological features were hilar adenitis 47 cases (39.16%), Paratracheal adenitis 20 cases (16.67%), Lymphangitis 40 cases (33.33%), Mottling 22 cases (18.33%), Consolidation 2 cases (1.67%), Collapse 1 case (0.83%), Pleural effusion 2 cases (1.67%), Pleural thickening 2 cases (1.67%), Cavitation 2 cases (1.67%).

Cough with or without expectoration 70 cases, radiological features were hilar adenitis 58 cases (48.33%), Paratracheal adenitis 26 cases (21.67%), Lymphangitis 40 cases (33.33%), Mottling 42 cases (35.0%), Consolidation 14 cases (11.67%), Collapse 8 cases (6.67%), Pleural effusion 8 cases (6.67%), Cavitation 6 cases (5.0%), Pleural thickening 2 cases (1.67%).

Failure to thrive 44 cases (36.67%), radiological features were - Hilar adenitis 25 cases (20.83%), Paratracheal adenitis 11 cases, (9.16%), Lymphangitis 8 cases (6.67%), Mottling 18 cases, (15.0%), Consolidation 2 cases (1.67%), Pleural effusion 3 cases (2.5%), Collapse 2 (1.67%), cavitation 2 (1.67%), Pleurant thickening 1 (0.83%).

Failure to thrive 44 cases (36.67%), radiological features were - Hilar adenitis 25 cases (20.83%), Paratracheal adenitis 11 cases (9.16%), Lymphangitis 8 cases (6.67%), Mottling 18 cases (15.0%), Consolidation 2 cases (1.67%), Pleural effusion 3 cases (2.5%), Collapse 2 (1.67%), cavitation 2 (1.67%), Pleurant thickening 1 (0.83%).

Breathlessness 27 cases (22.5%), radiological features were - Hilar adenitis 12 cases (10.0%), Paratracheal adenitis 7 cases (5.83%), Lymphangitis 11 cases (9.16%), Mottling 12 cases (10.0%), Consolidation 4 cases (3.33%), Pleural effusion 8 cases (6.67%), Collapse 8 cases (6.67%), Cavitation 3 cases (2.5%).

Recurrent respiratory tract infection 44 cases, radiological features were - Hilar adenitis 30 cases (25.0%), Paratracheal adenitis 12 cases (10.0%), Lymphangitis 20 cases (16.67%), Mottling 21 cases (17.5%).

Cervical adenitis 54 cases, radiological pictures were - Hilar adenitis 38 cases (31.67%), Paratracheal adenitis 15 cases (12.5%), Lymphangitis 26 cases (21.67%), Mottling 27 cases (22.5%), Consolidation 3 cases (2.5%), Collapse 2 cases (1.67%), Pleural thickening 2 (1.67%), Generalised adenitis 20 cases (16.67%), Radiological features were - Hilar adenitis 13 cases (10.83%), Paratracheal adenitis 8 cases (6.67%), Lymphangitis 6 cases (5.0%), Mottling 9 cases (7.5%), Consolidation 2 (1.67%), Collapse 1 (0.83%), Pleural effusion 2 (1.67%).

Hemoptysis - 4 cases radiological picture were Hilar adenitis 3 cases (2.5%), Lymphangitis 4 cases (3.33%), Mottling 3 cases (2.5%), Cavitation 4 cases (3.33%).

GIT symptoms 31 cases, radiological features were - Hilar adenitis 20 cases (16.67%), Paratracheal adenitis 10 cases (8.33%), Lymphangitis 12 cases (10.0%), Mottling 6 cases (5.0%).

TABLE - XII  
Showing clinico-radiological correlation

| Clinical features                     | No. of cases | Radiological presentation |                       |              |          |                |               |                  |                  |
|---------------------------------------|--------------|---------------------------|-----------------------|--------------|----------|----------------|---------------|------------------|------------------|
|                                       |              | Hilar adenitis            | Paratracheal adenitis | Lymphangitis | Mottling | Collapsed apse | Consolidation | Pleural effusion | Cavitated lesion |
| Pyrexia                               | 61           | 40                        | 26                    | 45           | 35       | -              | 4             | 2                | 4                |
| Pallor                                | 40           | 46                        | 20                    | 40           | 22       | 1              | -             | 2                | 2                |
| Cough with or without expectoration   | 70           | 58                        | 26                    | 40           | 22       | 8              | 14            | 8                | 6                |
| Failure to thrive                     | 44           | 25                        | 11                    | 12           | 18       | 2              | 2             | 3                | 2                |
| Breathlessness                        | 27           | 12                        | 7                     | 11           | 12       | 8              | 4             | 8                | 3                |
| Recurrent respiratory tract infection | 44           | 30                        | 12                    | 20           | 21       | -              | -             | -                | -                |
| Cervical adenitis                     | 54           | 38                        | 15                    | 26           | 27       | 2              | 3             | -                | -                |
| Generalised adenitis                  | 20           | 13                        | 8                     | 6            | 9        | 1              | 1             | 2                | -                |
| Hemoptysis                            | 4            | 3                         | -                     | 4            | 3        | -              | -             | -                | 4                |
| GIT symptoms                          | 31           | 20                        | 10                    | 12           | 6        | -              | -             | -                | -                |

Out of 120 cases the detailed radiological analysis noted were - Hilar adenitis in male 55 cases (45.83%) and female 33 cases (27.5%).

TABLE - XIII  
Showing radiological analysis of Hilar adenitis

| Age in years | Male |       | Female |      |
|--------------|------|-------|--------|------|
|              | No.  | %     | No.    | %    |
| 0 - 12       | 55   | 45.83 | 33     | 27.5 |

Out of 120 cases, the distribution of Hilar lymph node as per the side, age and sex-wise is 0-1 year male 2 cases (1.67%), right side 2 cases (1.67%), left lung 1 case bilateral (0.83%), female 2 cases right side (1.67%) 2 cases left side (1.67%) and 1 case bilateral (0.83%). 1-3 years age in male 11 cases right side (9.16%), 6 cases left side (5.0%), 7 cases bilateral (5.83%), in female 1 case right side (0.83%), 2 cases left side (1.67%), 3 cases bilateral (2.5%). 3-5 years age group in male 8 cases right side (6.67%), 7 cases in left side (5.83%), 3 cases bilateral (2.5%), in female 7 cases right side (5.83%), 1 case left side (0.83%) and 4 cases bilateral (3.33%). 5-10 years in male 5 cases right side (4.16%), 2 cases left side (1.67%) and 1 case bilateral (0.83%), in female 3 cases right side (2.5%), 2 cases left side (1.67%) and 5 cases bilateral (4.16%). 10-12 years 1 male case on right side (0.83%).



TABLE - XIV

Showing detailed radiological analysis age, sex-wise for Hilar adenitis.

| Sl. No. | Age in years | Sex | Right lung | Left lung | Bilateral |
|---------|--------------|-----|------------|-----------|-----------|
| 1.      | 0 - 1        | M   | 2          | -         | 1         |
|         |              | F   | 2          | 2         | 1         |
| 2.      | 1 - 3        | M   | 11         | 6         | 7         |
|         |              | F   | 1          | 2         | 3         |
| 3.      | 3 - 5        | M   | 8          | 7         | 3         |
|         |              | F   | 7          | 1         | 4         |
| 4.      | 5 - 10       | M   | 5          | 2         | 1         |
|         |              | F   | 3          | 2         | 5         |
| 5.      | 10 - 12      | M   | 1          | -         | -         |
|         |              | F   | -          | -         | -         |

Out of 120 cases lymphangitis with or without mottling observed in 0-12 years male 53 (44.16%), Female 32 (26.67%).

Table - XV

Showing sex-wise distribution of Lymphangitis

| Age in years | Male |       | Female |       |
|--------------|------|-------|--------|-------|
|              | No.  | %     | No.    | %     |
| 0 - 12       | 53   | 44.16 | 32     | 26.67 |

Out of 120 cases, lymphangitis with and without mottling detailed age and sex-wise were observed 0-1 year male right side 4 cases (3.33%), left side 0 case,

Bilateral 1 case (0.83%), female right side 2 cases (1.67%), left side 1 case (0.83%), bilateral 2 cases (1.67%). 1-3 years male right side 10 cases (8.33%), left side 5 cases (4.16%), bilateral 6 cases (5.0%), female right side 1 case (0.83%), left side 1 case (0.83%), bilateral 3 cases (2.5%). 3-5 years, male right side 6 cases (5.0%), left side 6 cases (5.0%), bilateral 6 cases (5.0%), female right side 5 cases (5.0%), left side 2 cases (1.67%), bilateral 3 cases (2.5%). 5-10 years male right side 4 cases (3.33%), left side 4 cases (3.33%), bilateral 1 case (0.83%), female right side 4 cases (3.33%), left side 2 cases (1.67%), bilateral 4 cases (3.33%). 10-12 years, male right side 1 case (0.83%).

TABLE - XVI

Showing radiologically lymphangitis with or without mottling.

| Sl. No. | Age in years | Sex | Right lung | Left lung | Bilateral |
|---------|--------------|-----|------------|-----------|-----------|
| 1.      | 0 - 1        | M   | 4          | 0         | 1         |
|         |              | F   | 2          | 1         | 2         |
| 2.      | 1 - 3        | M   | 10         | 5         | 6         |
|         |              | F   | 1          | 1         | 3         |
| 3.      | 3 - 5        | M   | 6          | 6         | 6         |
|         |              | F   | 5          | 2         | 3         |
| 4.      | 5 - 10       | M   | 4          | 4         | 1         |
|         |              | F   | 4          | 2         | 4         |
| 5.      | 10 - 12      | M   | 1          | -         | -         |
|         |              | F   | -          | -         | -         |

Out of 120 cases, consolidation was observed from 0-12 years. Male 10 cases (8.33%) & female 4 cases (3.33%).

TABLE - XVII

Showing sex-wise distribution of consolidation

| Age in years | Male |      | Female |      |
|--------------|------|------|--------|------|
|              | No.  | %    | No.    | %    |
| 0 - 12       | 10   | 8.33 | 4      | 3.33 |

Out of 120 cases age and sex-wise distribution of consolidation noted as 1-3 years male right side 4 cases (3.33%), left side 1 case (0.83%), female right side 1 case (0.83%). 3-5 years male right side 3 cases (2.5%), left side 2 cases (1.67%), female right side 1 case (0.83%). 5-10 years, female right side 1 case (0.83%), left side 1 case (0.83%).

TABLE - XVIII

Showing age-sex wise distribution of consolidation

| Sl. No. | Age in years | Sex | Right lung | Left lung | Bilateral |
|---------|--------------|-----|------------|-----------|-----------|
| 1.      | 0-1          | M   | -          | -         | -         |
|         |              | F   | -          | -         | -         |
| 2.      | 1-3          | M   | 4          | 1         | -         |
|         |              | F   | 1          | -         | -         |
| 3.      | 3-5          | M   | 3          | 2         | -         |
|         |              | F   | 1          | -         | -         |
| 4.      | 5-10         | M   | -          | -         | -         |
|         |              | F   | 1          | 1         | -         |
| 5.      | 10-12        | M   | -          | -         | -         |
|         |              | F   | -          | -         | -         |

Out of 120 cases collapse was noted from 0-12 years : male 6 cases (5.0%), Female 2 cases (1.67%).

TABLE - XIV

Showing age-sex wise distribution of Pleural effusion

| Age in year | Male |     | Female |      |
|-------------|------|-----|--------|------|
|             | No.  | %   | No.    | %    |
| 0-12        | 6    | 5.0 | 2      | 1.67 |



Out of 120 cases age and sex-wise distribution of collapse noted as 0-1 year male right side 1 case (0.83%), 1-3 years male left side 1 case (0.83%), 3-5 years male right side 2 cases (1.67%), female left side 1 case (0.83%), 5 - 10 years male right side 2 cases, (1.67%), female left side 1 case (0.83%).

TABLE - XX

Showing age and sex-wise distribution of Pleural effusion.

| S.I. No. | Age in years | Sex | Right side | Left side | Bilateral |
|----------|--------------|-----|------------|-----------|-----------|
| 1.       | 0 - 1        | M   | -          | 1         | -         |
|          |              | F   | -          | -         | -         |
| 2.       | 1 - 3        | M   | -          | 1         | -         |
|          |              | F   | -          | -         | -         |
| 3.       | 3 - 5        | M   | 2          | -         | -         |
|          |              | F   | -          | 1         | -         |
| 4.       | 5 - 10       | M   | 2          | -         | -         |
|          |              | F   | -          | 1         | -         |

Out of 120 cases pleural effusion was noted from 0-12 years : Male 4 case (3.33%), female 4 cases (3.33%).

TABLE - XXI

| Age in years | Male |      | Female |      |
|--------------|------|------|--------|------|
|              | No.  | %    | No.    | %    |
| 0 - 12       | 4    | 3.33 | 4      | 3.33 |

Out of 120 cases age sex-wise distribution of pleural effusion noted as - 1-3 years, male left side 1 case (0.83%), 3 - 5 years male right side 3 cases (2.5%), 5-10 years female right side 1 case (0.83%) left side 3 cases (2.5%).

TABLE - XXII

Showing age-sex wise distribution of pleural effusion

| Sl. No. | Age in year | Sex | Right side | Left side | Bilateral |
|---------|-------------|-----|------------|-----------|-----------|
| 1.      | 0 - 1       | M   | -          | -         | -         |
|         |             | F   | -          | -         | -         |
| 2.      | 1 - 3       | M   | -          | 1         | -         |
|         |             | F   | -          | -         | -         |
| 3.      | 3 - 5       | M   | 3          | -         | -         |
|         |             | F   | -          | -         | -         |
| 4.      | 5 - 10      | M   | -          | -         | -         |
|         |             | F   | 1          | 3         | -         |
| 5.      | 10 - 12     | M   | -          | -         | -         |
|         |             | F   | -          | -         | -         |

Out of 120 cases cavitation was observed from 0-12 years male 4 cases (3.33%) and female 2 cases (1.67%).

TABLE -XXIII

| Age in Years | Male |      | Female |      |
|--------------|------|------|--------|------|
|              | No.  | %    | No.    | %    |
| 0 - 12       | 4    | 3.33 | 2      | 1.67 |

TABLE - XXIV

Showing age-sex wise distribution of cavitation

| Sl. No. | Age in years | Sex | Right side | Left side | Bilateral |
|---------|--------------|-----|------------|-----------|-----------|
| 1.      | 0 - 1        | M   | -          | -         | -         |
|         |              | F   | -          | -         | -         |
| 2.      | 1 - 3        | M   | 1          | -         | -         |
|         |              | F   | -          | -         | -         |
| 3.      | 3 - 5        | M   | 3          | 1         | -         |
|         |              | F   | -          | -         | -         |
| 4.      | 5 - 10       | M   | -          | -         | -         |
|         |              | F   | 1          | 1         | -         |
| 5.      | 10 - 12      | M   | -          | -         | -         |
|         |              | F   | -          | -         | -         |

Out of 120 cases pleural chickening was observed as 0-12 years male - 3 cases (2.5%) and female 1 case (0.83%).

TABLE - XXV

Showing age-sex wise distribution of pleural thickening

| Sl. No. | Age in years | Sex | Right lung | Left lung | Bilateral |
|---------|--------------|-----|------------|-----------|-----------|
| 1.      | 0 - 1        | M   | -          | -         | -         |
|         |              | F   | -          | -         | -         |
| 2.      | 1 - 3        | M   | 1          | -         | -         |
|         |              | F   | -          | -         | -         |
| 3.      | 3 - 5        | M   | -          | 1         | -         |
|         |              | F   | 1          | -         | -         |
| 4.      | 5 - 10       | M   | -          | 1         | -         |
|         |              | F   | -          | -         | -         |
| 5.      | 10 - 12      | M   | -          | -         | -         |
|         |              | F   | -          | -         | -         |

INVESTIGATION :

Mantoux test was done in all 120 cases. All cases were positive. Size of induration was 10-15 mm. in 55 cases (45.83%), 15-20 mm. in 45 cases (37.5%), 20-25 mm. in 16 cases (13.33%), 25-30 mm. in 4 cases (3.33%).



TABLE - XXVI

Showing size of induration after 48-72 hours of injection

| No. of cases | Percentage | Size of induration |
|--------------|------------|--------------------|
| 55           | 45.83      | 10 - 15 mm.        |
| 45           | 37.50      | 15 - 20 mm.        |
| 16           | 13.33      | 20 - 25 mm.        |
| 4            | 3.33       | 25 - 30 mm.        |

Out of 120 cases routine investigation of 108 cases were done for TLC, DLC and ESR.

TABLE - XXVII

Showing total white blood cells count/cmm.

| Total white cell count cells/cmm. | No. of cases | Percentage |
|-----------------------------------|--------------|------------|
| 3000 - 5000                       | 19           | 15.80      |
| 5000 - 11000                      | 70           | 58.33      |
| 11000 - 15000                     | 19           | 15.80      |

Out of 120 cases ESR was done in 108 cases  
raised was observed in majority of cases.

TABLE - XXVIII

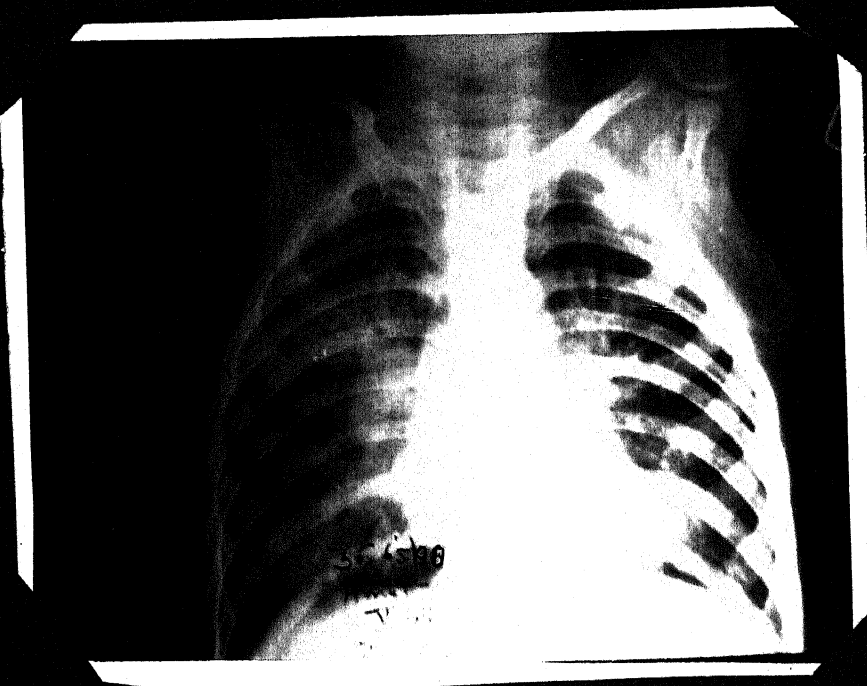
Showing E.S.R./Finit hour

| E.S.R.           | No. of cases | Percentage |
|------------------|--------------|------------|
| Upto 15 mm.      | 4            | 3.33       |
| 15 - 20 mm.      | 20           | 16.67      |
| 20 - 25 mm.      | 13           | 10.83      |
| 25 - 30 mm.      | 31           | 25.83      |
| More than 30 mm. | 40           | 33.33      |

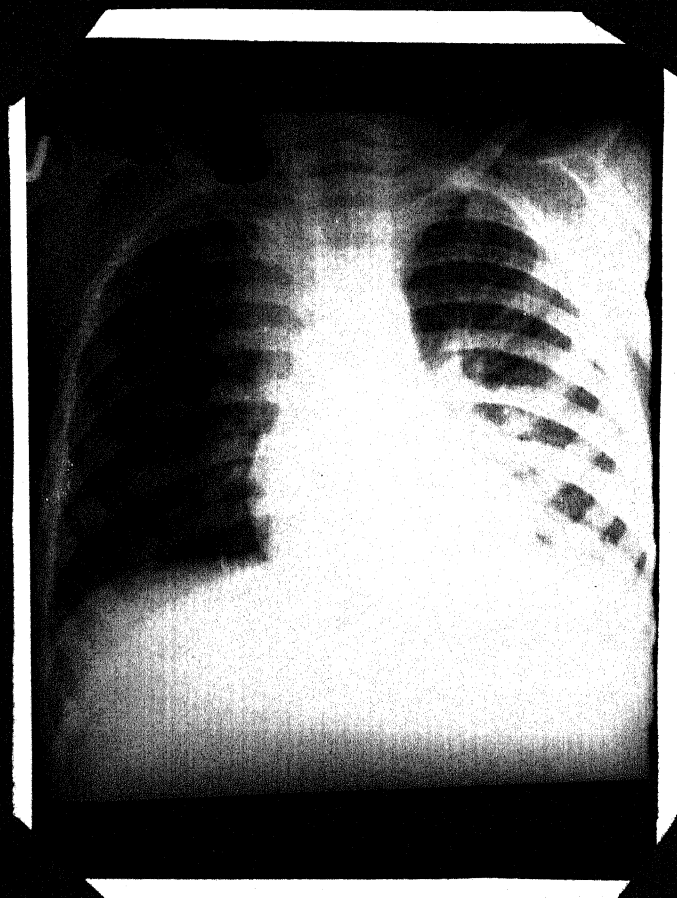
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Positive mantoux test in a case of  
primary tuberculosis

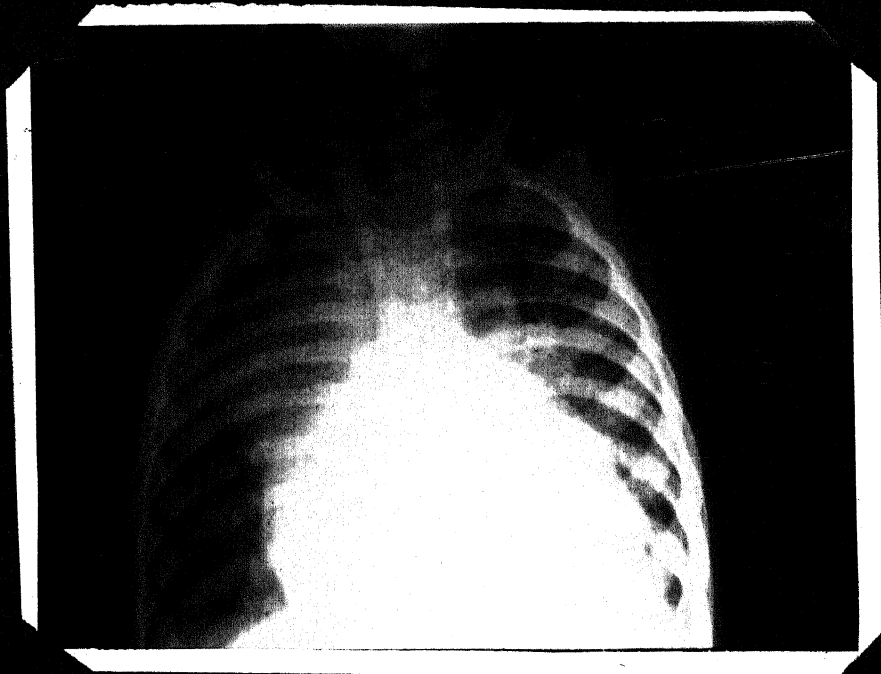


X-Ray chest P-A view , bilateral hilar adenitis, lymphadenitis and mottling.

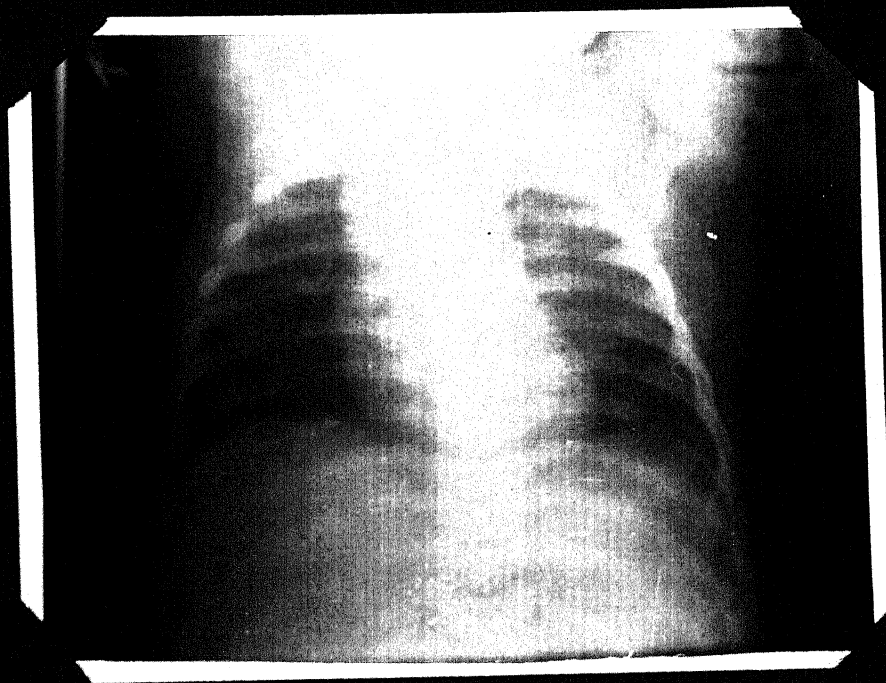


X-ray chest PA view, Rt. side paratracheal adenitis, bilateral hilar adenitis, Lt. side lymphangitis and mottling.

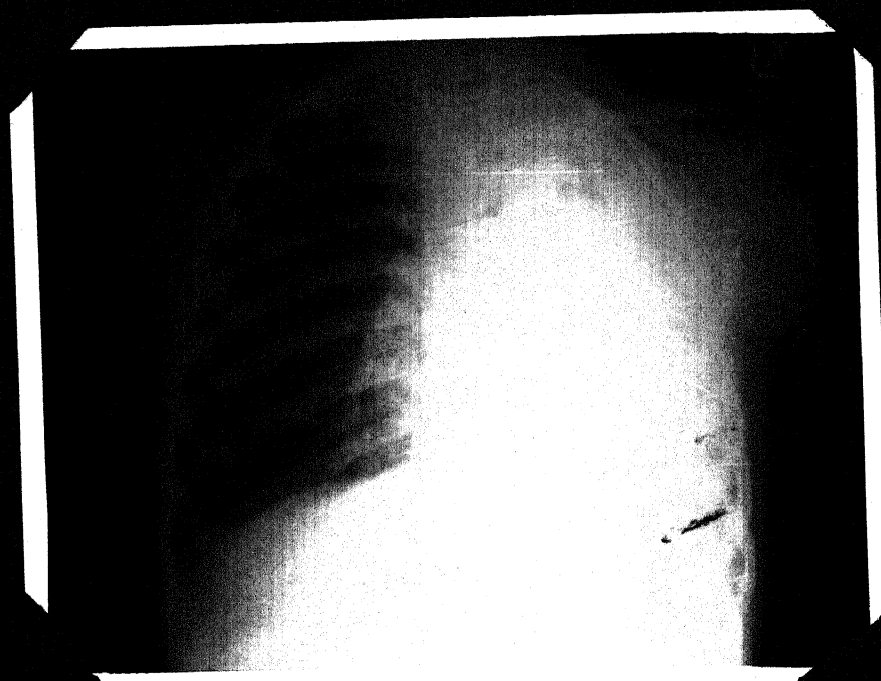




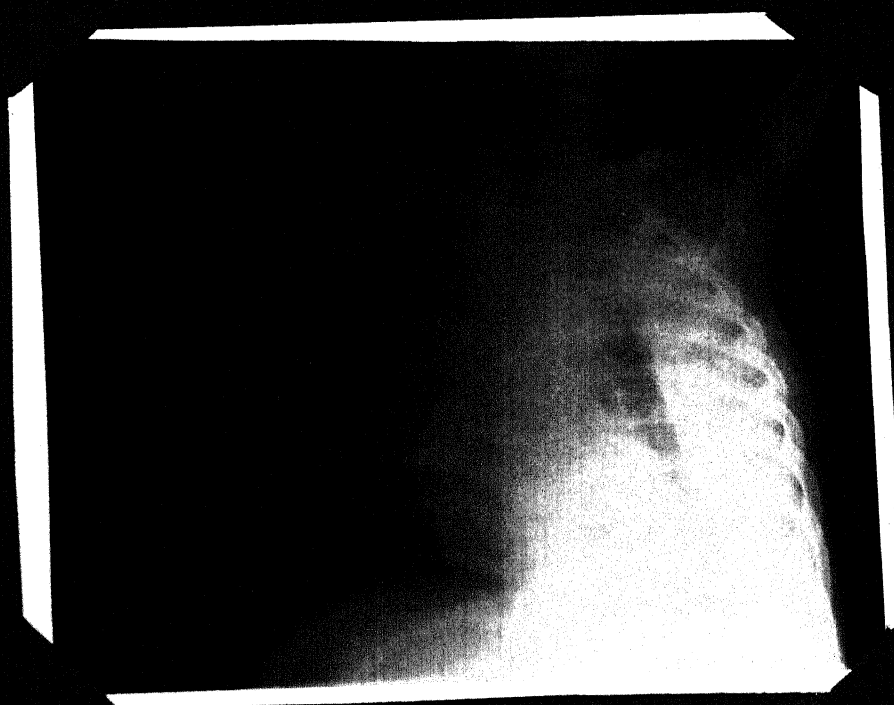
X-Ray chest PA view, bilateral hilar adenitis  
lymphangitis and mottling with cardiomegaly.



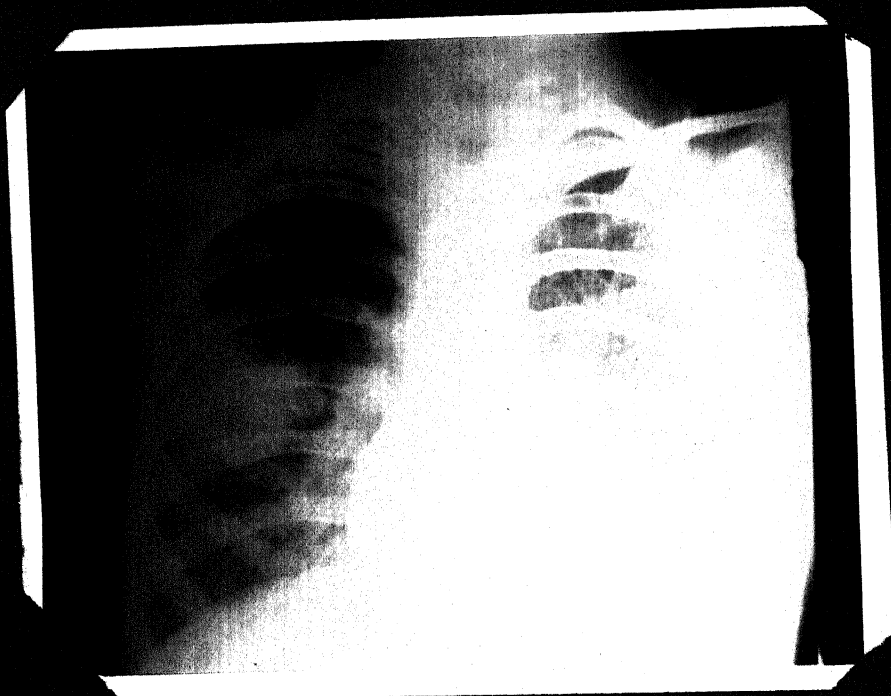
X-Ray chest PA view, Rt. side paratracheal  
adenitis.



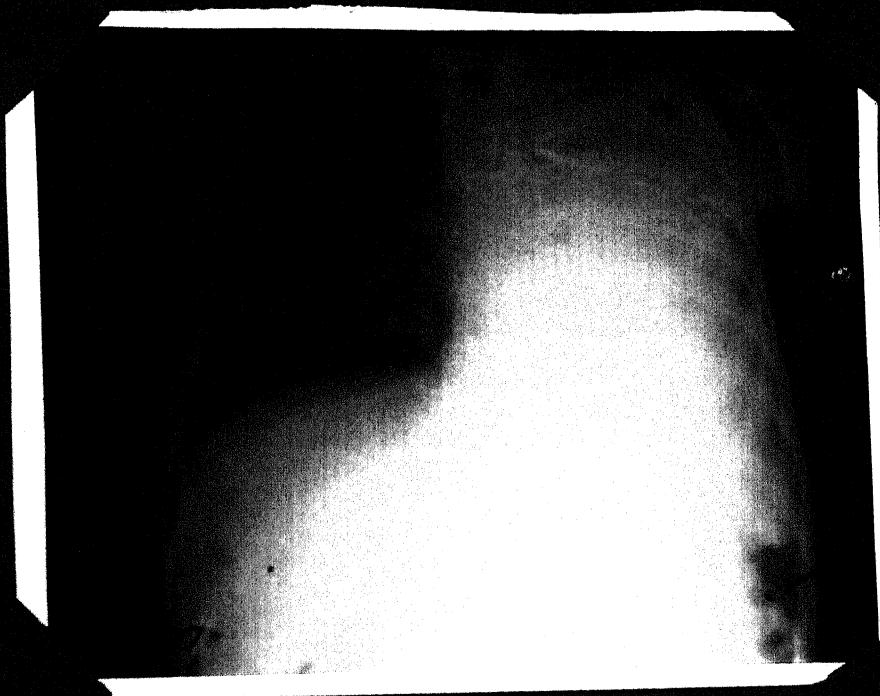
X-Ray chest PA view, consolidation of  
Lt. lung with collapse.



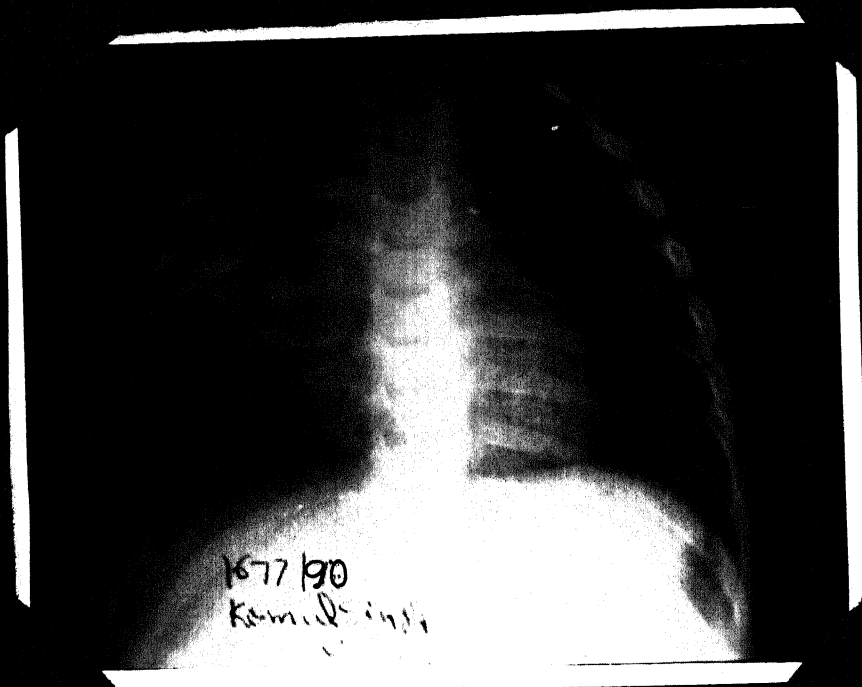
X-Ray chest PA view, Rt. side hilar adenitis,  
consolidation of Lt. lung with effusion.



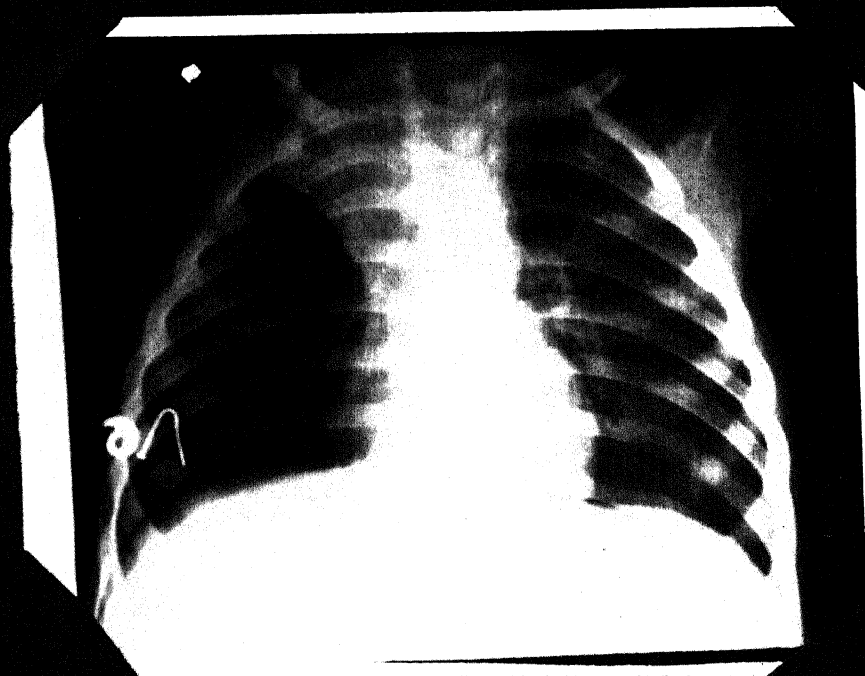
X-Ray chest PA-view, bilateral hilar adenitis, consolidation on Rt. side with mottling and lymphangitis on Lt. side.



X-Ray chest PA view, consolidation of Lt. lung.

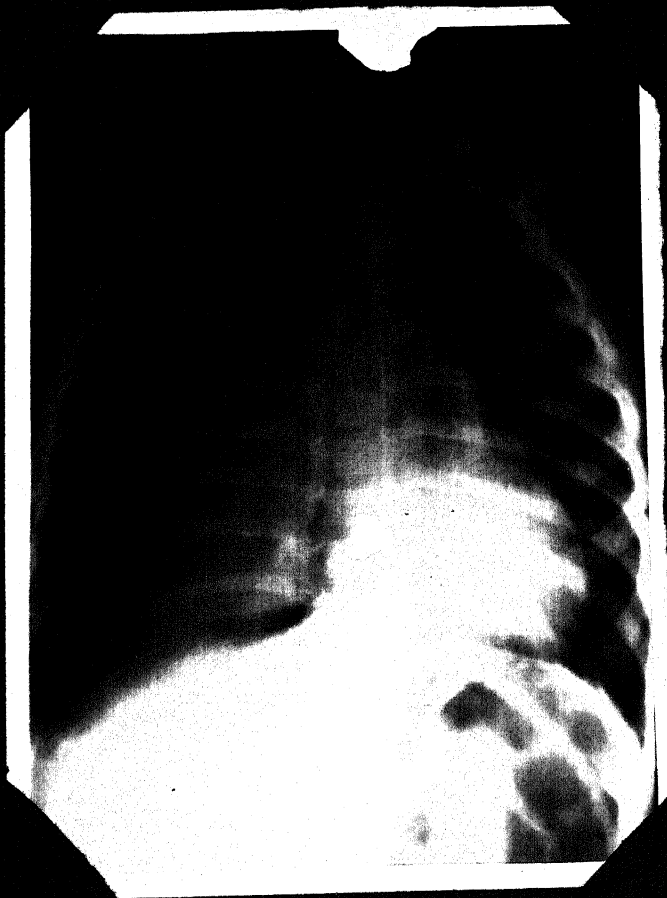


X-ray chest PA view, Infiltration and cavitation in Rt. middle zone with hilar lymphadenitis.

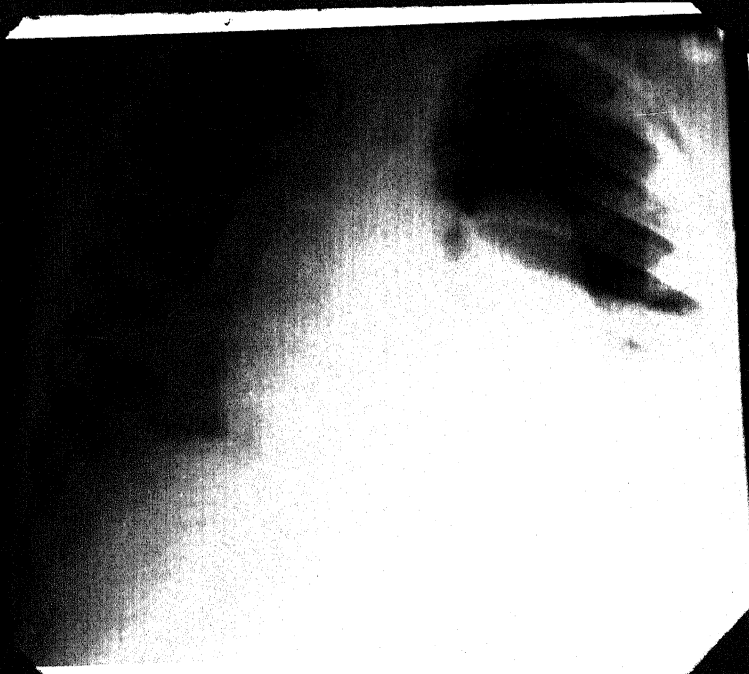


X-ray chest PA view, collapse of Rt. upper lobe.





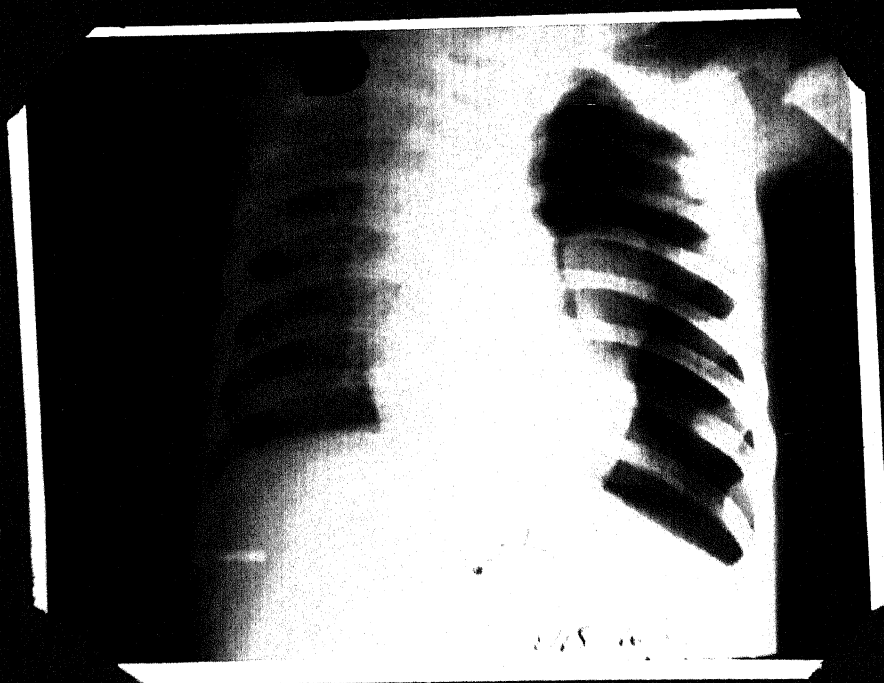
X-Ray chest PA view, Pneumothorax with collapse of Rt.lung.



X-Ray chest PA view, hydropneumothorax on Lt.side with collapse of Lt.lung.



X-Ray chest PA view, absorption  
collapse of Rt. lung.



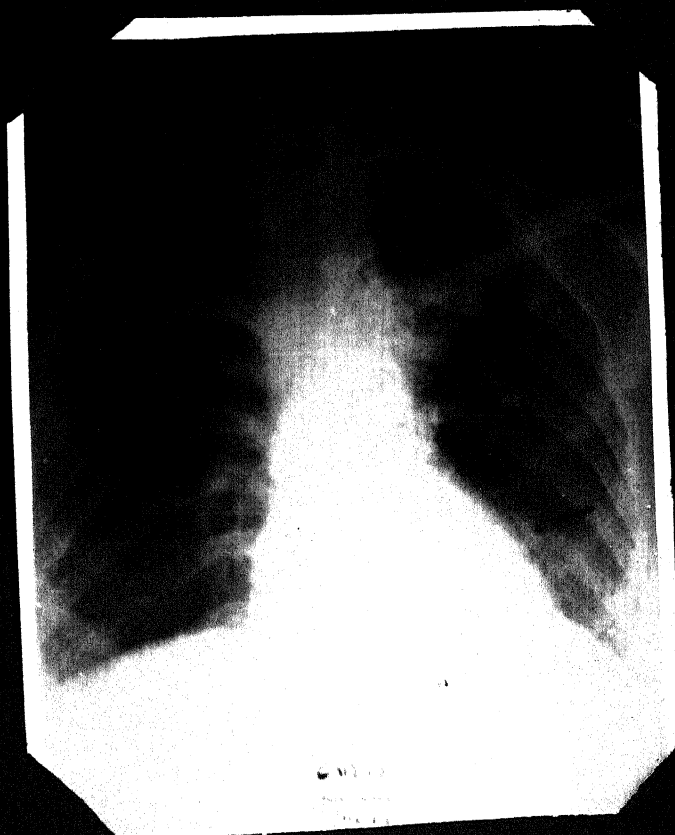
X-Ray chest PA view, Mediastinal adenitis  
on Rt. side with pneumothorax on Lt. side.



X-ray chest PA view, Rt. side paratracheal adenitis and consolidation in Rt. upper lobe.



X-ray chest PA view, bilateral hilar adenitis and lymphangitis.



X-Ray chest PA view. calcified patches in  
Lt. middle zone with calcified glands.



DISCUSSION

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## DISCUSSION

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## DISCUSSION

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Tuberculosis is one of the important public health problem of India has several special features. Several epidemiological fields works in last few decades have revealed, these features. Further elucidation are still needed for this problem.

The present study comprised of 120 children of 0-12 years of age having a positive relevant clinical findings, mantoux positive and radiologically positive cases were taken into account.

A mantoux test with 1 T.U. P.P. Dof RT 23 timer 80 strength was performed in all 120 cases. Children showing tuberculin reaction more than 10 mm. induration in transvers diameter after 72 hours were considered positive cases.

In present study, 120 cases of 0-12 years of age were taken. About 6% of children of families taken in study did not co-operate even though they were offered free services, so they were dropped out. As regarding mangoux test about 3% of cases did not turn up after hours, they also were dropped out.

Radiological examination of children despite of repeated attempts skiagram of about 1% children were

technically defective due to faulty technique and developing solution, they could not be interpreted, they therefore, were discarded.

In present study the incidence of pulmonary tuberculosis was found to be highest 33.33% in age group 1-3 years, and second highest 31.67% in children of 3-5 years age group. Kotaiah (1958) published his study of primary pulmonary tuberculosis in 195 cases at Visakhapatnam. He observed that highest incidence 88.20% of tuberculosis in children higher age group 10-15 years, and 11.80% in below three years of age, 98% of his cases were mantoux positive. Manchanda et al, (1966) reported 215 cases from Amritsar, and found higher incidence in 5-10 years age group. Prakash et al (1963) observed 273 tuberculin positive children at Lucknow and found the incidence of 10.8%, 32.6% and 40.7% among 0-3 years, 3-7 years and 7-12 years. In 1966 Ramchandran and Furnayyan from Tanjavur presented an analytical study of 365 children. They found that the maximum incidence was 46.8% amongst 0-2 years age group.

The incidence of primary tuberculosis was found to be higher in comparison to other worker. This could be because of the observed higher prevalence of disease in

general in the present study. Secondly the criteria particularly about the selection of cases may be different in studies of other workers. However, this is confirmatory with observation in our present study and also by different workers that the younger child (below 5 years of age) are more prone to develop disease.

Higher pulmonary tuberculosis under 5 years can be explained with on the basis of their early susceptibility to harbour the infection. Other contributory factor could be close proximity and sharing of food stuff with the index cases, who in most of cases was either of parents. Secondly they are more prone to develop disease because of poor immunity to tuberculosis (P.M. Udani and K.A. Krishnamurthy and J. Vishwanathan, 1972).

The percentage of male and female children in our study was 64.17% and 35.83%. The sex percentage was not significant in the prevalence of disease but higher percentage of cases were noted in male child. The higher percentage in males is higher age group, was due to their greater degree of outdoor activities and thus having a greater chance of being exposed to infection. Secondly, they may have come in contrast with some unknown, undiagnosed open case of tuberculosis regarding which history could not be obtained.



Generally it is believed that tuberculosis is a problem of poor people. It holds true to a large extent due to difference in standard of living, nutritional status, overcrowding, poor general hygiene etc. In the present study, most of the children were from the low socio-economic group. High prevalence of infection was found in children of low socio-economic status. Karen (1964) also observed that poor socio-economic children are slightly more afflicted than well to do families.

According to Benjamin (1957), the standard of living is directly proportional to incidence to tuberculosis. Pamra & Mathur (1968) in a survey among the Delhi civil servant reported that the prevalence and incidence of tuberculosis in low socio-economic status was nearly 3 times more than middle income group.

The explanation for higher prevalence of tuberculosis in children from low socio-economic status may be given on basis of aforesaid factors. Other contributory factor could be higher prevalence of tuberculosis in adult population. In poor community, including ignorance, lack of child care and various environmental factors.

In our study we did mantoux test in 188 clinically suspected cases of primary tuberculosis, 166 were turned up after 72 hours, and only 132 were positive. Out of 132 only

in 120 cases, there was radiologically evidence of primary pulmonary tuberculosis. In our present study radiological finding were as follows :-

Hilar adenitis 73.33%, paratracheal adenitis 41.67%, lymphangitis 70.83%, mottling 54.17%, consolidation 11.67%, collapse 6.67%, pleural effusion 6.67%, cavitation 5% and pleural thickening 3.33%. As forementioned most common radiological presentation of primary tuberculosis in our study was Hilar adenitis followed by Lamphangitis and Mottling.

A study was conducted by Ramchandran (1976) in which he studied 3000 for radiological evaluation of primary tuberculosis. The criteria for registration was -

1. Suspicious symptoms including repeated respiratory tract infection, chronic diarrhoea and failure to thrive.
2. Mantoux positivity.
3. Radiological positivity in chest X-ray.

Out of 3000 children registered, 1630 X-rays only were available for study. Radiological finding of these X-ray as follows -

No X-ray finding in 263 (16.13%), intrathoracic lymphnodes only 527 (32.33%), intrathoracic lymphnode with

infiltration 309 (18.95%), lymphanodes with multiple infiltration 86 (5.27%), miliary tuberculosis 86 (5.27%), others 164 (10.06%).

Bently and Grybowski (1954) reported that out of 317 children with uncomplicated primary complex 115 had a paranchymal focus and 202 had only lymphadenopathy.

In a study of 538 children (Walker, 1958), primary complex were seen in 280 cases (mottling with hilar adenitis), primary cavitation in 5, broncho-pneumonia in 3 and miliary tuberculosis 60 cases.

Manchanda et al (1966) reported their observations on 225 children, 43 cases were found to have primaty complex, 86 cases were found to have lymphanopathy, collapse were in 4 cases, consolidation in 11 cases, bronchopneumonia in 2 cases, pleural effusion in 5 cases, middle lobe syndrome in 2 and cavitation in 1 patient.

Dinglay (1966) conducted a study and found that out of 500 cases of primary tuberculosis 260 had glandular involvement and 28 cases had primary complex, consolidation in 170 cases, collapse in 37 cases, pleural effusion in 56 and calcification in 5 cases.

Table - I

Comparison of Radiological picture in Primary Tuberculosis in Children

| Sl. No. | Workers                      | Radiological features |                         |                 |            |                   |                                |                            |
|---------|------------------------------|-----------------------|-------------------------|-----------------|------------|-------------------|--------------------------------|----------------------------|
|         |                              | Mottling %            | Glandular involvement % | Consolidation % | Collapse % | Cavit-<br>ation % | Miliary<br>tuber-<br>culosis % | *Other<br>Pl. Eff. %<br>** |
| 1.      | Ramchandran<br>(1976)        | 18.95                 | 56.56                   | 8.93            | -          | -                 | -                              | 10.06 1.53                 |
| 2.      | Bantli & Grybowski<br>(1954) | 36.28                 | 63.72                   | -               | -          | -                 | -                              | -                          |
| 3.      | Walker (1958)                | 30.26                 | 52.04                   | -               | -          | 0.90              | 11.15                          | 5.90 -                     |
| 4.      | Manchanda (1966)             | 19.11                 | 38.22                   | 4.88            | 1.77       | 0.40              | -                              | 1.70 2.20                  |
| 5.      | Dingley (1966)               | 5.60                  | 52.00                   | 34.00           | 7.40       | -                 | -                              | 1.00 11.20                 |
| 6.      | Sharma, K.P. (1968)          | 38.04                 | 78.26                   | 19.56           | -          | -                 | -                              | 47.82 7.50                 |
| 7.      | Present study                | 54.16                 | 73.32                   | 11.66           | 6.66       | 5.00              | -                              | 70.83 6.66                 |

\* Other includes following radiological features:- lymphangitis, pleural thickening, interlobular effusion, middle lung syndrome, bronchopneumonia, calcification etc.

\*\* Pl. Eff. - Pleural effusion.



In our study most common radiological presentation of primary tuberculosis was glandular enlargement 73.33%. It was more common on right side 55% and left side 40%. It is slightly higher than other studies.

Ramchandran (1976) found only glandular enlargement in his study was 51.28%. The various groups of lymphanodes involvement reported as follows - Superior mediastinal adenitis 21 cases (13.19%), right hilar adenitis 165 cases (10.12%), left hilar adenitis (2.20%), bilateral hilar adenitis 53 cases (3.25%) and all glands 58 cases (3.55%).

We found only glandular enlargement (hilar adenitis) in 35 cases (29.17%) in which 25 were in right lung (20.83%), 10 were in left lung (8.33%), and bilateral 10 cases (8.33%).

Right side glandular enlargement is more common in primary tuberculosis. In assessment of hilar shadows specially on the right side many more pitfalls are possible eg. a child with acute infection or recurring from upper and lower zone respiratory tract infection or from measles or whooping cough or those with heart lesion and left to right shunt, the left hilar nodes usually escapes alteration because of their reterocardiac location but slightly over exposed film will bring them up more clearly.

Ramchandran (1976) found in his study primary complex (lymphadenopathy with paranchymal infiltration) in 309 cases (18.95%). In majority of cases infiltration was in right upper lobe and lymphnode was often superior mediastinal or right hilar. Milar (1963) reported that in his series of 525 children with a primary complex only in 16%. Walker (1958) in his study found 280 cases of primary complex out of 538 cases (52.04%). Manchanda et al (1966) reported 43 cases of primary complex out of 225 children (19.11%).

We found in our study that out of 120 cases typical primary complex was seen only in 43 cases (35.83%) out of which 38 were in right lung, 15 were in left lung and 14 cases were bilateral. Zone wise distribution of primary complex was as follows -

Table - II

Showing zone wise distribution of primary complex

| Zone-wise   | Right lung | Left lung |
|-------------|------------|-----------|
| Upper zone  | 20         | 8         |
| Middle zone | 16         | 5         |
| Lower zone  | 2          | 2         |

Similar to other worker we found in our study that primary complex is more common in right side in comparison to left side and oftenly involves right upper lobe in comparison to middle and lower lobe. The parenchymal infiltration is usually single seen any where in the lung fields and on a few occasion infiltration are seen one side where as lymphadenitis is on the opposite side. We consider opacities which are not in time with or related to a bronchial or vascular marking as suggestive of tuberculous infiltration.

We, in our study, found radiographic linear shadows in between mottling and lymphadenopathy. It was actually lymphangitis. In 85 cases (70.83%) out of 120 cases we noted lymphangitis. In many cases lymphangitis helps to locate the Ghon's foccus and many cases Ghon's foccus was not seen and only lymphangitis was seen. The incidence of lymphangitis in our study was much higher in comparison to other studies. K.P. Sharma (1988) found lymphangitis denoted as diversion of blood vessels in 31 cases out of 92 cases.

In our present study we observed consolidation in 14 cases, most of them were on right side 10 cases and 4 cases were on left side. We considered it tubercular consolidation after administration of antibiotics for 7

days to see whether consolidation clears. Out of 14 cases consolidation, 10 were in right lung and 4 were in left lung. Ramchandran (1976) found 134 cases of consolidation (8.93%) and mostly was in right lung. Manchand (1966) reported 11 cases of consolidation out of 225 cases and mostly were in right lung. Dinglay (1966) observed consolidation in 170 cases out of 500 cases. K.P. Sharma (1988) observed 18 cases (19.56%) of consolidation mostly on right side.

We observed pleural effusion in 8 cases (6.67%), equally distributed in both sides. It was more common in male and 1-3 years children were more involved. Ramchandran (1976), observed 23 cases (1.53%) of pleural effusion. He observed no predilection for occurrence of effusion on any one side effusion. Manchanda (1966) reported 5 cases (2.22%) of pleural effusion and mostly were on right side. Dinglay (1966) reported 56 cases (11.2%) of pleural effusion and mostly were on right side.

Ramchandran (1976) observed that pleural effusion on X-ray examination is described as a curved shadow, it is more often a horizontal line in children due to fluid in inter-lober septum or to co-existing segmental lesion. Quite often the pleural effusion was revealed by a



vertical line following the attachment of parital pleural to the apex of the lung.

We noted collapse, including compression collapse and absorption collapse, in 8 cases (6.67%) more in male 6 cases and 2 in female. They were equally distributed. Collapse was more common in 3-10 years of age group children, out of 8 cases 3 cases were of compression collapse due to hydropneumothorax and 5 cases were of absorption collapse. In absorption collapse mostly were of upper lobe collapse on right side. Manchanda (1966) reported of 4 cases (1.77%) of collapse out of 225 cases. Dinglay (1966) observed collapse in 37 (7.4%) out of 500 cases and mostly were on right side.

We observed cavitation in 6 cases (5%), 4 were in right lung and 2 were in left lung. Cavitation was more common in 3-10 years male child and mostly found in middle and upper lobe of right lung. Manchanda (1966) observed one case (0.4%) of cavitation out of 225 cases of primary tuberculosis.

We observed pleural thickening in only 4 cases (3.33%). It has more common in male and equally distributed on both sides, 2 cases in right lung and 2 cases in left lung.

A Govindan and R. Narmeda (1976) conducted a study and observed that in 1,140 cases out of 1,500 the pleural thickening was seen giving an overall percentage of 76%. Further 945 out of 1,140 cases (i.e. 63%) had a positive mantoux test and in only 195 cases (13%) the mantoux test was negative as shown in table - III.

Table - III

Showing result of Mantoux Test

| Total<br>no. of<br>cases | Mantoux<br>result | No. of cases<br>with pleural<br>thickening | No. of cases<br>without pleural<br>thickening | %   |
|--------------------------|-------------------|--------------------------------------------|-----------------------------------------------|-----|
| 1500                     | Positive          | 945                                        | -                                             | 63% |
| 1500                     | Negative          | 195                                        | -                                             | 13% |
| 1500                     | Positive          | -                                          | 360                                           | 24% |

We conducted mantoux test in 188 children clinically suspected primary tuberculosis. Only 166 were turned up after 72 hours and out of 166 only 132 (79.51%) were positive i.e. induration was more than 10 mm after 72 hours. All the 166 children were examined radiologically for evidence of primary tuberculosis. Out of 132 cases mantoux positive in 120 (90.90%) cases, there were evidences of primary tuberculosis in one or other way. In 12 cases where there is no radiological evidence of P.T., they must be suffering from GIT tuberculosis or

other form of tuberculosis. A.Govindan & R. Narmada (1976) conducted a study, they did mantoux test in 1500 clinically suspects children and observed mantoux positive in 1305 cases (87%) N.R. Bhandari (1984) conducted a study comparison between B.C.G. test and mantoux test, observed that in proved 165 cases only in 78 cases (47.27%) mantoux test was positive.

Negative mantoux test in radiologically proved primary tuberculosis can be explained as these children were either marasmic or under steroid therapy. These may faulty technique causing negative test.

In our study we observed that incidence of primary tuberculosis is much higher than study conducted done by other workers. It can be explained very well as in Bundelkhand mostly people are uneducated, living in very poor environment, have low socio-economic status. Hilar adenitis in the most common radiological presentation of primary tuberculosis. The percentage of hilar adenitis, mottling and lymphangitis is higher in our study in comparison to other worker. The difference may be due to selection of cases and mode of study may be different in their study.

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## SUMMARY & CONCLUSION

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### SUMMARY & CONCLUSION

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The present study entitled "Radiological evaluation" of primary tuberculosis in Mantoux test positive children" was conducted in department of Pediatrics and Radiology of M.L.B. Medical College, Jhansi. The study comprised of 120 children of 0-12 years of age. Out of 120 cases, 0-1 year 12 cases (10.0%), 1-3 years 40 cases (33.33%), 3-5 years 38 cases (31.67%), 5-10 years 28 cases (23.33%) and 10-12 years 2 cases (1.07%).

The criteria of selection of cases were as follows :-

1. Positive history suggestive of primary tuberculosis.
2. Mantoux Test positivity, i.e. more than 10 mm after 72 hours.
3. Radiological evidence of primary tuberculosis in chest X-ray.

Detailed history of present illness, past illness and family history was taken from parents. Complete physical examination was performed in every case with especial emphasis to respiratory examination and lymphnode examination. In all cases mantoux test was done with 5 TU of PPD, RT 23 Tween 80 and their X-ray chest PA was done. Observations were tabulated and data analysed.

In present study prevalence of primary tuberculosis was found to higher in 1-3 years of age group (33.33%) and second highest in 3-5 years age group (31.67%).

Male dominated in this study with (64.17%) and female (35.83%). Prevalence of primary tuberculosis has higher in low socio-economic children in comparison to higher group children.

The most common radiological presentation of primary tuberculosis was hilar adenitis (73.33%) followed by lymphangitis (70.83%), mottling (54.17%), Paratracheal (41.67%), consolidation (11.67%), collapse (6.67%), pleural effusion (6.67%), cavitation (5%) and pleural thickening (33.33%).

Right side lung is more commonly involved. Right side hilar adenitis was observed in (34.17%), while left side (18.33%) and bilateral (20.83%). Paratracheal adenitis right side (21.67%), left side (10.83%), bilateral (9.17%). Lymphangitis right side (31.67%), left side (17.5%) and bilateral (21.67%), mottling right side (30.83%), left side (11.67%) and bilateral (11.67%), consolidation right side (8.33%), left side (3.33%). Collapse right side (3.33%), left side (3.33%), pleural effusion right side (3.33%), left side (3.33%). Cavitation right side

(3.33%), left side (1.67%), pleural thickening right side (1.67%), left side (1.67%).

Typical primary complex was seen in (35.83%) cases, out of which (25.83% in right lung, (6.67%) in left lung and bilateral in (11.67%) cases. Hilar lymphadenitis alone was seen in (29.17%) cases, out of which (16.67%) on right side, (4.17%) cases left side and bilateral (8.33%) cases.

Although we included only mantoux positive children but we conducted mantoux test in 188 cases having positive history of primary complex. Only 166 were turned up after 72 hours. In 166 only 132 (79.51%) were positive. Out of 132 positive mantoux test only in 120 (90.90%), we found radiological features of primary tuberculosis.

Following conclusions were made from present study:

1. Prevalence of primary tuberculosis is higher in 1-3 years of age group in comparison to other age group.
2. Children from low socio-economic status are more prone to primary tuberculosis.
3. Male children suffer more primary tuberculosis in comparison to female children.
4. High incidence of primary tuberculosis occur in anaemic and malnourished children.

5. The main radiological finding in primary tuberculosis is hilar adenitis followed by lymphangitis and mottling.
6. Right side involvement is higher in comparison to left side, and as far as zone is concern upper zone is more involved.

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# **BIBLIOGRAPHY**

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BIBLIOGRAPHY

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1. Agarwal, V.K.; Kumar, P and Srivastava, N.N. (1966) : Indian Pediatrics, 3 : 364.
2. Balkrishnan, S. and Sharma, S. (1962) : Ind. J. Ch. Health, 11 : 365.
3. Basu, A.K.; Madan, M.S. and Rao, M.S. (1959) : Ind. J. Ch. Health, 8 : 476.
4. Barucha, P.E. and Rodrigues, P.F. (1961) : Ind. J. Ch. Health, 10 : 552.
5. Benjamin, B. (1951) : Tubercle, 82.
6. Benjamin, P.V. (1959) : A Sample Survey I.C.M.R., 1955-56, Special Report Series no. 34.
7. Bentley, F.J. (1958) : Tavistock Square, London.
8. Brailey, M.E. and Hardy, J.B. (1958) : Quoted from "Tuberculosis in children", Miller, F.J.W.
9. Briggs, B.; Illingworth, R.S. and Lorber, J. (1955) : Lancet, 1 : 263.
10. British Tuberculosis Association (1959) : Tubercle, 40 : 317.
11. Caffey, J. (1956) : Pediatric X-ray diagnosis.

12. Caffey John (1967) : Paediatric X-ray diagnosis.  
5th edition.
13. Chatterjee, D. (1957) : J.I.M.A., 29 : 180.
14. Cohen, S. (1957) : Dis. Chest, 2 : 207.
15. Datta, T. and Sen, K. (1980) : Diagnosis of  
childhood tuberculosis at BCG Workshop, New Delhi.
16. Davis, P.D.B. (1960) : Tubercle (Lond. Supple.)  
1 : 40.
17. Davis, S.F.; Finely, S.C. and Hase, W.K. (1960) :  
J. Ped., 57 : 221.
18. Dhar, H.N. and Mishra, S.P. (1973) : Indian J. of  
Tuberculosis.
19. Faridi, A.J. (1944) : J.I.M.A., Nov., 26.
20. Flynn, M.P. and Joyce, J.C. (1954) : Tubercle, 35 :  
270.
21. Frimdot-Moller (1960) : Bull. World Health Org.,  
22 : 61.
22. Frimdot J. Moller (1960) : A community-wide tuber-  
culosis study in a South Indian Rural Population.
23. Frimdot-Moller (1962) : Tubercle, 42 : 88.

24. Furcolow, M.L. (1941) : Quoted from "Tuberculosis in Children", Miller, F.J.W.
25. Gefel, A.; Pruzanski, W.; Altman, R. and Lorant, T. (1963) : Acta. Tub. Scand., II : 309.
26. Ghon, A. (1912) : Quoted from "Tuberculosis in Children". Miller, F.J.W.
27. Gordon, W.L. (1959) : Tubercle, 40 : 446.
28. Gothi, G.D. Nair and Rao, K.P. (1968) : Tuberculosis in rural population of South India.
29. Haef, F. (1959) : Recent advances in Resp. Tub., 164.
30. Hartzberg (1942) : Quoted by M.M. Singh.
31. Hugedon (1946) : Arch. Dis. Chil., 21 : 121.
32. I.C.M.R. (1955-58) : Tuberculosis in India.
33. I.C.M.R. / W.H.O. Report (1980) : Meeting at Delhi, April / May, 1980.
34. Koch, R. (1960) : Tubercle, 41 : 123.
35. Kotaiah, P. (1958) : Ind. J. Ped., 25 : 241.
36. Lester, C.E. (1958) : Am. Rev. Res. Dis., 78 : 399.
37. Macleod, H.M. and Rose, J.K. (1962) : Tubercle, 43 : 151.



38. Maha, G.E. (1963) : Clinical Med., 70 : 761.
39. Manchanda, S.S.; Bawa, Y.S. and Bhatia, J.L. (1958) : Ind. J. Chil. Health, 7 : 938.
40. Mascher, Willis (1951) : Am. Rev. Tub., 63 : 501.
41. Manual of B.C.G. Technician (1960) : Govt. of India, W.H.O. and U.N.I.C.E.F. (Jt. Pub.).
42. Medical Research Council of Gr. Britain (1962) : Lancet, 1 : 582.
43. Miller, F.J.W. and Seal, R.M.E. (1963) : Tuberculosis in children.
44. Miller, F.J.W.; Seal, R.M.E. and Taylor, M.D. (1963) :
45. Medd, W.E. (1969) : BCG test in diagnosis of tuberculosis. Lancet.
46. Negpal, D.R. (1967) : District Tuberculosis Control Programmes. Indian J. Tuberculosis.
47. Nelson, W.E. (1959) : Editorial, J. Ped., 55 : 541.
48. Nelson, W.E. (1964) : Text Book of Ped., 8th Ed.
49. Nyboe, J. (1960) : Bull. W.H.O., 225.
50. Onstead, C. and Small Piece, V. (1957) : B.M.J. 2 : 1437.

51. Palmer, C.E. (1953) : B.M.J., 1 : 363.
52. Palmer, C.E.; Edward, L.B. and Edward, E.C. (1959) : J. of Ped., 55 : 413.
53. Parrot (1876) : Quoted from Miller, F.J.W.
54. Prakash, R. and Sharma, N.L. (1963) : Thesis, M.D., Lucknow University.
55. Prasad, B.G. (1961) : Bri. J. of Dis. of Chest, 55 : 169.
56. Proceeding of BCG Workshop held in Oct. (1980).
57. Raj Narain (1963) : Tuberculosis Prevalence Survey in Tumkur District. Indian. J. Tuberculosis.
58. Ramachandran, R.S. and Purneyyan, S. (1966) : Ind. Ped., 1 : 45.
59. Ram Krishnan, C.V. (1962) : I.J. of Tub., 11 : 50.
60. Ranke (1916) : Quoted by Holmes- seldors.
61. Rich, A.R. and McCordock, H.A. (1929) : Quoted from Miller, F.J.W.
62. Reddi, Y.R.; Rajeshwaramma and Rao, P.S. (1964) : Ind. Ped., 1 : 45.
63. Roy, S.K. (1962) : Ind. Med. J., 56 : 163.
64. Seibert, F.B. and Munday, B. (1934) : Am. Rev. Tuber., 25 : 274.

65. Sarin, L.R.; Samuel, K.C. and Bhargava, R.K. (1957) :  
Am. Rev. Tuber. and Resp. Dis., 76 : 410.
66. Sekulich, M. (1955) : Tuberculosis Classification -  
Pathogenesis and Management.
67. Shanks, S.C. and Karley, P. (1973) : Tuberculosis in  
childhood. A text book of X-ray diagnosis. 4th ed.  
London, H.K. Lewis & Co.
68. Sikand, B.K. (1962) : Indian J. of Public Health.
69. Singh, M.M. (1953) : Thesis for M.D. (Tub.)  
Lucknow University.
70. Smith, C.B. (1951) : Am. J. of Ch. Path., 21 : 674.
71. Thompson, B.C. (1949) : B.M.J., 2 : 841.
72. Thompson, B.C. (1957) : Am. Rev. Res. Dis., 75, 6 :  
886.
73. Udani, P.M. (1960) : Ind. J. Chil. Health, 9 : 565.
74. Udani, P.M. (1962) : Journal S.N. Medical College,  
Agra.
75. Udani, P.M. (1962) : Ind. J. Chil. Health, 11 : 372.
76. Udani, P.M. (1968) : Tuberculosis in children.  
Paediatric Clinic, India.
77. Udani, P.M.; Parik Usha and Shah, P.M. (1971) : BCG  
test in tuberculosis. Indian Paediatrics.

78. Udani, P.M.; Usha, S. Bhat and Bhave, S.K. (1976) : Epidemiology, morbidity, mortality and control programme. Indian Paediatrics, 5.
79. Udani, P.M. (1980) : Key note. BCG Workshop, New Delhi.
80. Ukil, A.C. (1937) : Quoted by M.M. Singh.
81. Ukil, A.C. (1930) : J.Am. Res., 17 : 821.
82. Ustvedt, H.S. (1949) : Reaction to B.C.G.
83. Walker, C.H.M. (1955) : Lancet, 1 : 218.
84. Wallgren, A.(1939) : Quoted by Bentley et al.
85. Wallgren, A.(1948) : Tubercle, 29 : 245.
86. Wallgren, A.(1950) : Tuberculosis and other problems of Pediatrics, Baltimore, Williams and Wilkins.
87. Wagle, M.M. (1966) : Ped. Cli. Ind., 1 : 15.
88. W.H.O. (1962) : Bull. of Int. Union Against Tub., 32, 1-88.
89. Zitrin, C.M.; Lincoln, E.M. and Melly, E. (1963) : Abstract of Med., 33, 13 (Art. 15).
90. Zitrin, C.M. (1960) : Am. Rev. Res. Assoc., 121 : 266.

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